ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 52 JUNE, 1943 No. 2

XXIII

A HISTORICAL SURVEY OF THE STRUCTURE AND FUNCTION OF THE COCHLEA

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The perception of external stimuli is a problem which has always intrigued philosophers and scientists. This is especially true of the problem, "How do we hear?" Of all the organs for sense perception the ear is the best protected, the most delicate and structurally the most complicated. New facts regarding its structure are still being found; pathology and disfunction of the ear still baffle the otologist; and the conflicting interpretations as to how we hear still belong to the realm of theory. More scientists have their names attached to the structure of this organ than to any other part of the body and the varied interpretations of its functions form a bewildering maze.

The earliest theory of sound perception is the theory of "aer implantus" propounded by Aristotle in the fourth century, B. C. According to this Greek concept, for the perception of sound it was necessary to have a cavity in the head filled with air to respond to the impulses originating in the outer air. Thus the popular almost ridiculous statement, "It passes in one ear and out the other," has a historic background and does in a certain sense signify how far re-

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moved the common concept is from the intricate structure and the function of the auditory organ.

According to Shambaugh,[88] Perrault in 1680 had the idea that in the ear cavity there existed a vibrating mechanism which responded to tone waves, and Duverney in 1683 was the first to liken the cochlea to a musical instrument which responds in different parts to different tones according to the principles of physical resonance. He believed that the lamina spiralis was the vibrating mechanism and, because this bony plate is broader in the basal coil and becomes gradually narrower toward the apex, he located perception of low tones in the basal coil and the high tones in the apex.

The finding of fluid in the internal ear by Cotugno in 1760, and later the finding of many delicate structures, led to constantly changing views as to how external sounds reach the sensorium.

The general structure of the outer, middle and inner ear were known by the old anatomical masters but the discoveries of the finer anatomical structures of the cochlea, on which the various theories of hearing have been built, belong to the past century.

According to Rutherford,[86] Newton in 1704, although an opponent of the undulatory theory of light, nevertheless suggested that light induces vibrations in the retina which are transmitted to the brain and there give rise to color sensations that differ according to the length of the incoming vibrations.

In 1824 Huschke[60] found in the cochlea the ridge of tall cells among which the nerves terminated and he considered it as a sensory papilla similar to the sensory papilla in the ampulla of the semicircular canal. He also described the stria vascularis and the tall cells covering the limbus. Thus three structures in the ear come to bear his name: (1) Huschke's papilla; (2) Huschke's teeth;[61] and (3) stria vascularis of Huschke.

According to Rutherford,[86] Johannes Müller in 1826 propounded the theory of "specific energies" or "specific activities." Although this concept is similar to Newton's, yet he wrote about Newton's idea as follows: "such a hypothesis if at all tenable would find first application in accounting for the different sensations of which a single sense is susceptible; for example, in explaining how the sensorium receives the different impressions of red, yellow and blue and of acute and grave tones," but suggests that, "it is only with this application that the theory is worthy of regard." These wise words of Müller, however, were set aside and such men as Natanson in 1844, according to Rutherford[86] and Helmholtz[48, 49] in 1863 extended Müllers hypothe-

sis of specific activities. Müller's theory of specific activities thus modified is clearly stated by Hermann[54] in the following words:

> "It is necessary to ascribe to the stimulations of different nerve fibers the different qualities of any given sensation; for example, the sensation of red and blue, of high and low tones, and hence to suppose that the specific energy of certain fibers aids in the perception of red, while that of others aids the perception of blue, for if it were not so we would be compelled to assume that one and the same nerve fiber is capable of states of excitement that differ in quality, an assumption hitherto unsupported by facts. There must, therefore, be at least as many sensory fibers as there are simple qualities of sensation."

This theory of specific activities of nerve fibers is the basic principle of the resonance theory.

The real impetus to interpretation of the functions of the ear came when Corti[16] in 1851 gave a detailed description of the papilla of Huschke which has since then been known as the organ of Corti. The following structures bear his name: (1) organ of Corti, (2) pillar of Corti, (3) tunnel of Corti, (4) hair cells (outer) of Corti, and (5) membrane of Corti (tectorial membrane). During the same decade in which Corti published his work on the ear other scientists contributed to our knowledge of this organ. Deiter[24] described the inner hair cells and the supporting cells of these hair cells. It is interesting to note that in the recent textbooks Deiter's name is associated with the supporting cells of the hair cells but not with the inner hair cells. When and by whom this erroneous interpretation was given we have not been able to learn.

In 1858 Claudius[13] described the cells lining the outer sulcus spiralis and these cells have come to be known as the cells of Claudius. He also showed that the basilar membrane is a thin membrane where it spans the tunnel of Corti. This finding plays an important role in several of the later theories of hearing. Claudius, according to Hensen,[51] held that sound impulses were transmitted through the tympanum secondarium to the cochlea, a view which was accepted by many, even by Hensen as late as 1863.

Although Corti and others had described in some detail the basilar membrane with its attached organ of Corti, they had failed to recognize the various spaces within the cochlea. The common concept, even when Helmholtz formulated his theory in 1863, was that there were two spaces separated incompletely from each other by a membrane, the basilar membrane. The two spaces were known as the vestibular gallery (scala vestibuli) and the tympanic gallery (scala tympani). According to Hensen,[51] Reichert thought these two spaces communicated with each other at the hamulus and at the vestibule.

Reissner's membrane was not recognized and what is scala media was considered part of the scala vestibuli.

In the same year, 1851, when Corti[16] described the organ which bears his name, Reissner[82] in a dissertation at Dorpat presented evidence that in the cochlea there existed a special canal, the "canalis cochlearis." Three years later in 1854 no one had openly considered his findings. He, therefore, published another paper[83] in which he described the otic vesicle in fetuses and showed how this vesicle constricts to form three parts: (1) the recessus labyrinthi, which up to this time was falsely considered the same as the aquaductus cochlearis and was called the aquaductus vestibuli; (2) the vestibular portion with the semicircular canals; and (3) the ductus cochlearis (scala media), which he showed was originally the only space within the cochlea. He said that one end of this canal was closed and the other opened into the cavities of the vestibule. This canal was first called "canalis cochlearis" by Reissner. He showed quite correctly that this canal was at first closely surrounded by cartilage, which gradually receded and thus gave rise to the two perilymph cavities, the scala tympani and the scala vestibuli. He described the walls of the canal, the basilar membrane, the stria vascularis and the vestibular membrane. To the latter the name Reissner's membrane was given by Kölliker.[65] In spite of these careful observations the canalis cochlearis (scala media) was not generally recognized until some time after Kölliker[65] in 1861 had verified the findings of Reissner. Kölliker at first thought the canal was closed at both ends.

Claudius[14] in 1856 and Loewenberg[70] as late as 1864 and Henle[50] in 1866 thought that the tectorial membrane was attached at the modiolus and lay over the teeth of Huschke and over the organ of Corti and was attached to the outer wall or spiral ligament. These men must have seen a collapsed Reissner's membrane and thought it part of the tectorial membrane.

During the decade following 1851 the knowledge of the detailed anatomy of the cochlea was rapidly taking shape, but many structures were as yet imperfectly understood. This imperfection, however, did not deter the scientific minds from trying to associate function with any new structure and thus theories of "How we hear" soon came into being.

THE RESONANCE THEORY (HELMHOLTZ'S EARLY CONCEPT)

Corti's analysis of the organ which bears his name was one of the major factors which induced Helmholtz[48] to speculate on the function of these structures and this led to the formulation of the

well-known resonance theory. Starting with the basic premise of Müller's theory of specific activities, that specific nerve fibers can transmit only specific sensations, he ascribed to specific parts of the organ of Corti the duty of picking up certain stimuli and transmitting them to definite nerve fibers. To formulate such a theory it required a mind that was versed not only in anatomy and physiology but also in physics. Helmholtz, more than anyone else at his time, had these qualifications. He had been trained in the medical sciences, obtained his medical degree, taught for several years and then devoted himself to the teaching of physics.

When Helmholtz first advanced his theory, the structure of the ear was still poorly understood and this led to certain misconceptions. One of these was the idea of Claudius that the auditory impulses from the middle ear were transmitted to the inner ear via the secondary tympanic membrane. Helmholtz carefully studied the middle ear structures and described the ligaments of the malleus and incus. He concluded that the lever arrangement of the middle ear ossicles was such that a movement of small force but great amplitude at the tympanic membrane was transformed to a movement of great force and small amplitude at the oval window, where he considered the impulse to enter the inner ear. Let me quote his own words:

"In this transference of the vibrations of air into the labyrinth it is to be observed that though the particles of air themselves have a comparatively large amplitude of vibration, yet their density is so small that they have no very great moment of inertia, and consequently when their motion is impeded by the drumskin of the ear, they are not capable of presenting much resistance to such an impediment or of exerting any sensible pressure against it. The fluid in the labyrinth, on the other hand, is much denser and heavier than the air in the auditory passage, and for moving it rapidly backwards and forwards as in sonorous oscillations, a far greater exertion of pressure is required than was necessary for the air in the auditory passage. On the other hand the amplitude of the vibrations performed by the fluid in the labyrinth are relatively very small, and extremely minute vibrations will in this case suffice to give a vibratory motion to the terminations and appendages of the nerves, which lie on the very limits of microscopic vision.

"The mechanical problem which the apparatus within the drum of the ear had to solve, was to transform a motion of great amplitude and little force, such as impinges on the drumskin, into a motion of small amplitude and great force, such as had to be communicated to the fluid in the labyrinth."

Another misconception which Helmholtz did *not* correct was that there were only two scala in the cochlea as is gathered from his own words:

"When the drumskin is driven inwards by increased pressure of air in the auditory passage, it also forces the auditory ossicles inwards, as already explained, and as a consequence the foot of the stirrup penetrates deeper into the oval window. The

fluid of the labyrinth, being surrounded in all other places by firm bony walls, has only one means of escape . . . the round window with its yielding membrane. To reach it, the fluid of the labyrinth must either pass through the helicotrema, the narrow opening at the vertex of the cochlea, flowing over from the vestibule gallery into the drum gallery, or, as it would probably not have sufficient time to do this in the case of sonorous vibrations, press the *membranous partition* of the cochlea against the drum gallery. The converse action must take place when the air in the auditory passage is rarefied."

Again Helmholtz knew little about the structure of the membranous partition (basilar membrane) and ascribed no special function to it except that it moved with the incoming vibrations and that this movement somehow brought into resonance certain parts of the organ of Corti. He at first put great emphasis on the pillars of Corti and also on the otoliths. He considered these pillars or strings to be set into vibration as sympathetic resonators and to be possible nerve terminals. He also discussed the otoliths at some length and suggested that this auditory sand might act as small hammers to strike on the strings. It appeared that he was not clear as to the role this auditory sand played but in his later fourth edition he still considers it, for he says:

"If we maintain the hypothesis, that every nervous fibre hears in its own peculiar pitch, we should have to conclude that the vibrating parts of the ear which convey these sensations of the highest tones to the ear, are much less sharply defined in their capabilities of resonance, than those for deeper tones. This means that they lose any motion excited in them comparatively soon, and are also comparatively more easily brought into the state of motion necessary for sensation. This last assumption must be made, because for parts which are so strongly damped, the possibility of adding together many separate impulses is very limited, and the construction of the auditory ciliae in the little bags of the otoliths seem to be more suited for this purpose than that of the shortest fibres of the basilar membrane. If this hypothesis is confirmed we should have to regard the auditory ciliae as the bearers of squeaking, hissing, chirping, crackling sensations of sound, and to consider their reaction as differing only in degree from that of the cochlear fibres."

For the perception of tone, however, he placed all emphasis on the pillars of Corti. This early concept of Helmholtz is summarized by Rutherford[86] as follows:

"How are we enabled to appreciate differences of pitch? As we all know, Helmholtz supposed that the same nerve cell cannot give us two different sensations even of the pitch of tone and therefore a separate nerve fiber and cell must be impressed to enable us to hear tones differing by, it may be, only the fraction of a vibration. This was his extension of the doctrine of specified activities to explain the different qualities of tone sensation. To render his theory feasible, he supposed that the cochlea must contain an immense number of minute resonators capable of severally responding by sympathetic vibration to all the tones he can hear. Each cochlear resonator was supposed to stimulate its own special nerve fiber and this in turn its

own cell in the brain. When he advanced that theory in 1862 the structure of Corti's organ was so imperfectly understood that Corti's pillars were supposed to be the nerve terminals. They increased very slightly in length from base to apex of cochlea (from 0.06 mm. at the base to 0.1 mm. at the apex), yet Helmholtz deemed that small difference sufficient for his theory."

Helmholtz's functional interpretations in their original form were soon challenged. Hasse's[47] important work (in 1870) on the cochlea in birds and amphibians showed that these animals have no pillars of Corti and yet they are capable of perceiving tones. These animals, however, do have hair cells and the tectorial membrane. Thus the hearing in these animals at least could not be dependent on the pillars of Corti.

Another very important publication which greatly influenced the subsequent interpretations of Helmholtz was that of Victor Hensen[51] in 1863. The additions to our knowledge of the cochlea which Hensen presented need only be listed here. He described (1) the hair of the hair cells as "stabechen" (rod-like structures); (2) the ductus reuniens; (3) the tall supporting cells outside the outer hair cells which bear his name; (4) the stripe of Hensen, a thickened band on the underside of the tectorial membrane; and (5) the basilar membrane which he showed was narrow in the basal turn and gradually became wider toward the apex of the cochlea. He says "I must insist, contrary to the present concept which probably dates back to Corti, that the membrane gradually widens from its root on upwards." He shows in a diagram that at the basal end it is about .04125 mm. wide, while at the hamulus it is .495 mm. wide. (6) He supported Claudius in the belief that the membrane is thin under the tunnel of Corti; (7) he described the basilar membrane as being made up of radial fibers and (8) he correctly described the aquaductus cochlearis. Reichert had stated that the scala vestibuli and scala tympani communicate at the hamulus and in the vestibule. Hensen correctly described the relationship of these two scalae.

HENSEN'S THEORY OF HEARING

Hensen[51] had his own idea as to the role played by the different parts of the cochlea in hearing. Here are his own words:

"I cannot withstand the temptation to present my idea of tone perception. I consider it not lucky or correct enough to place much emphasis on it but it offers a basis which may be of interest to the reader.

"It has been emphasized by Claudius[13] that the 'Tympanium secondarium' is the mechanism through which sounds are transmitted to the cochlea. The position of the shortest radix of the Basilar membrane which lies directly near the round window, the gradual broadening of the Basilar membrane and the delicacy of it

below the Papilla were reasons which forced me to accept the interpretation of Claudius, which previously, without any special reason, I did not lean to. When segments of the Basilar membrane, depending on their width, are brought in movement by means of incoming tones, then necessarily the papilla (Organ of Corti) must also move. The membrane of Corti (tectorial membrane) on the contrary cannot be influenced by such movement because it is attached only on those cells which are supported by the lamina ossea. Thus the 'Stabchen' (stiff hair) are more loosely or more firmly pressed (first those of the outer cells)—against the mass of Corti's membrane. The question is how through this reduced or increased pressure on the hair a sensation can be elicited."

He further states that the terminal plate of Corti's cells easily separates from the cells. This plate with the hair, therefore, seems to him as an independent structure which simply overlies Corti's cells. He then says, "If now the Corti cell is an end ganglion then it may be expected that the rarefication and compression of its protoplasm might readily give rise to a nerve impulse."

The work of Hasse and Hensen greatly influenced Helmholtz and as a result his book on *The Sensation of Tone* passed through a number of editions. In the preface of his third edition as given in the second English edition[49] he writes:

"The present Third Edition has been much more altered in some parts than the second. Thus in the sixth chapter I have been able to make use of the new physiological and anatomical researches on the ear. This has led to a modification of my view of the action of Corti's arches. Again, it appears that the peculiar articulation between the auditory ossicles called 'hammer' and 'anvil' might easily cause within the ear itself the formation of harmonic upper partial tones for simple tones which are sounded loudly."

His fourth edition appeared in 1877 and presents his views as they have come down to us. The generalized accounts by various reviewers of Helmholtz's theory which have come down to us do not always give us the true import of his own thoughts. For the sake of preserving accuracy of his concept, I will try to tell his story by quoting selected passages from his book. The following quotations are taken from the second English edition[49] revised to conform to the fourth German edition of 1877.

THE RESONANCE OR HARP THEORY

Helmholtz's basic concepts and reasons are clearly presented in the following passages:

"The ear does not distinguish the different forms of waves in themselves, as the eye distinguishes the different vibrational curves. The ear must be said rather to decompose every wave form into simpler elements according to a definite law. It then receives a sensation from each of these simpler elements as from an harmonious tone. By trained attention the ear is able to become conscious of each of these

simpler tones separately. And what the ear distinguishes as different qualities of tone are only different combinations of these simpler sensations.

"This analysis of compound into simple pendular vibrations is an astonishing property of the ear. The reader must bear in mind that when we apply the term 'compound' to the vibrations produced by a single musical instrument, the 'composition' has no existence except for our auditory perceptions, or for mathematical theory. In reality, the motion of the particles of the air is not at all compound, it is quite simple, flowing from a single source. When we turn to external nature for an analogue of such an analysis of periodical motions into simple motions, we find none but the phenomena of sympathetic vibration. In reality if we suppose the dampers of a pianoforte to be raised, and allow any musical tone to impinge powerfully on its sounding board, we bring a set of strings into sympathetic vibration, namely all those strings, and only those which correspond with the simple tones contained in the given musical tone. Here, then, we have, by a purely mechanical process, a resolution of air waves precisely similar to that performed by the ear. The air wave, quite simple in itself, brings a certain number of strings into sympathetic vibration, and the sympathetic vibration of these strings depends on the same law as the sensation of harmonic upper partial tones in the ear.

"Now suppose we were able to connect every string of a piano with a nervous fibre in such a manner that this fibre would be excited and experience a sensation every time the string vibrated. Then every musical tone which impinged on the instrument would excite, as we know to be really the case in the ear, a series of sensations exactly corresponding to the pendular vibrations into which the original motion of the air had to be resolved. By this means, then, the existence of each partial tone would be exactly so perceived, as it really is perceived by the ear. The sensations of simple tones of different pitch would under the supposed conditions fall to the lot of different nervous fibres, and hence be produced quite separately, and independently of each other.

"Now, as a matter of fact, later microscopic discoveries respecting the internal construction of the ear, lead to the hypothesis, that arrangements exist in the ear similar to those which we have imagined. The end of every fibre of the auditory nerve is connected with small elastic parts, which we cannot but assume to be set in sympathetic vibration by the waves of sound.

"The terminal expansions of these nerves, as I have already mentioned, are connected with very small elastic appendages, which appear adapted to excite the nerves by their vibrations."

In this fourth edition of his book he follows very closely his earlier editions in his anatomical descriptions and places considerable emphasis on the rods of Corti. After describing the sensory papilla of the ampulla, he writes:

"The construction of the cochlea is much more complex. The nerve fibres enter through the axis or modiolus of the cochlea into the bony part of the partition, and then come on to the membranous part. Where they reach this, peculiar formations were discovered quite recently (1851) by the Marchese Corti, and have been named after him. On these the nerves terminate.

"Among these, Corti's arches are relatively the most solid formations. The series of these contiguous arches consist of two series of rods or fibres, an internal and an external.

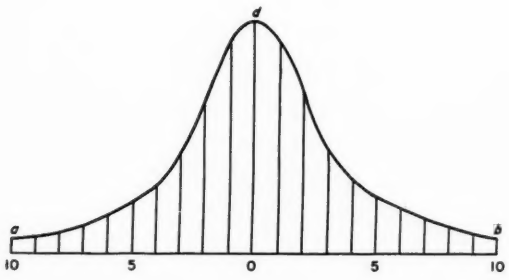

Fig. 1.—Helmholtz's illustration to show that one fiber *od* is set into strong vibration to a given tone but that a few other fibers on either side may vibrate but to a much smaller degree. The resonance area is quite specific and tapers off very rapidly.

"The fibres of the first series arise from the inner margin of the membrane, which can be relatively little agitated, but the fibres of the second series are attached nearly in the middle of the membrane, and this is precisely the place where its vibrations will have the greatest excursions. When the pressure of the fluid in the drum gallery of the labyrinth is increased by driving the foot of the stirrup against the oval window, the membrane at the base of the arches will sink downwards, the fibres of the second series be more tightly stretched, and perhaps the corresponding places of the fibres of the first series be bent a little downwards. It does not, however, seem probable that the fibers of the first series themselves move to any great extent for their lateral connections are strong enough to make them hang together in masses, like a membrane, when they have been released from their attachment in anatomical preparations. On reviewing the whole arrangement, there can be no doubt that Corti's organ is an apparatus adapted for receiving the vibrations of the membrana basilaris, and for vibrating of itself, but our present knowledge is not sufficient to determine with accuracy the manner in which these vibrations take place. For this purpose we require to estimate the stability of the several parts and the degree of tension and flexibility, with more precision than can be deduced from such observations as have hitherto been made on isolated parts, as they casually group themselves under the microscope.

"Hence when we hereafter speak of individual parts of the ear vibrating sympathetically with a determinate tone, we mean that they are set into strongest motion by that tone, but are also set into vibration less strongly by tones of nearly the same pitch, and that this sympathetic vibration is still sensible for the interval of a Semitone."

Thus far Helmholtz seems somewhat indefinite as to the function he assigns to the various structures of the cochlea. He still seems to lean to his earlier view that the pillars or rods of Corti are of prime importance but allows that all the structures in the organ of Corti "partly elastic, partly firm" may be put into vibration (Fig. 1). In the following line he continues in a similar mood: "Now we cannot

precisely ascertain what parts of the ear actually vibrate sympathetically with individual tones."

Here he leaves out his earlier statement regarding the role played by the "particles of auditory sand" but substitutes the following:

"We can only conjecture what they are, at present, in the case of human beings and mammals. The whole construction of the partition of the cochlea, and of Corti's arches which rest upon it, appears most suited for executing independent vibrations. We do not need to require of them the power of continuing their vibrations for a long time without assistance."

In this passage it should be noted that he speaks of the "partition of the cochlea." He seems still to ignore the other partition, namely Reissner's membrane. The cochlear duct or scala media is not mentioned by Helmholtz.

In the following paragraphs he then seems to lead up to the transition from his earlier to his later views:

"But if these formations are to serve for distinguishing tones of a different pitch, and if tones of different pitch are to be equally well perceived in all parts of the scale, the elastic formations in the cochlea, which are connected with different nerve fibers must be differently tuned, and their proper tones must form a regularly progressive series of degrees through the whole extent of musical scale.

"According to the recent anatomical researches of V. Hensen and C. Hasse, it is probably the breadth of the membrana basilaris in the cochlea, which determines the tuning. At its commencement opposite the oval window, it is comparatively narrow, and it continually increases in width as it approaches the apex of the cochlea.

Then suddenly he seems to have made up his mind for he writes:

"In the first (German) edition of this book (1862), which was written at a time when the more delicate anatomy of the cochlea was just beginning to be developed, I supposed that the different degrees of stiffness and tension in Corti's rods themselves might furnish the reason of their different tuning. By Hensen's measures of the breadth of the membrana basilaris[51] and Hasse's proof that Corti's rods are absent in birds and amphibia, far more definite foundations for forming a judgment have been furnished, than I then possessed.

"Hence it follows, as Henle has also proved, that the greatest increase of breadth falls on the outer zone of basilar membrane, beyond the line of the attachment of the rods. This increases from 0.023 mm. (equals .000905 inches) to 20.41 mm. (equals .016142 inches) or nearly twenty-fold.

"It has already been mentioned that the membrana basilaris of the cochlea breaks easily in the radial direction, but that its radial fibres have considerable tenacity. This seems to me to furnish a very important mechanical relation, namely that this membrane in its natural connection admits of being tightly stretched in the transverse direction from the modiolus to the outer wall of the cochlea, but can have only little tension in the direction of its length, because it could not resist a strong pull in this direction.

"Now the mathematical theory of the vibration of a membrane with different tensions in different directions shows that it behaves very differently from a mem-

brane which has the same tension in all directions. On a latter, vibrations produced in one part, spread uniformly in all directions, and hence if the tension were uniform it would be impossible to set one part of the basilar membrane in vibration without producing nearly as strong vibrations (disregarding individual nodal lines) in all other parts of the membrane.

"But if the tension in direction of its length is infinitesimally small in comparison with the tension in direction of the breadth, then the radial fibres of the basilar membrane may be approximately regarded as forming a system of stretched strings, and the membranous connection as only serving to give a fulcrum to the pressure of the fluid against these strings. In that case the laws of their motion would be the same as if every individual string moved independently of all others, and obeyed, by itself, the influence of the periodically alternating pressure of the fluid of the labyrinth contained in the vestibule gallery. Consequently any exciting tone would set that part of the membrane into sympathetic vibration, for which the proper tone of one of its radial fibres that are stretched and loaded with the various appendages already described which corresponds most nearly with the exciting tone; and thence the vibrations will extend with rapidly diminishing strength on to the adjacent parts of the membrane.

"Under these circumstances the parts of the membrane in unison of higher tones must be looked for near the round window, and those with the deeper, near the vertex of the cochlea, as Hensen also concluded from his measurements. That such short strings should be capable of corresponding with such deep tones, must be explained by their being loaded in the basilar membrane with all kinds of solid formations; the fluid of both galleries in the cochlea must also be considered as weighting the membrane, because it cannot move without a kind of wave motion in that fluid.

"The observations of Hasse show that Corti's arches do not exist in the cochlea of birds and amphibia, although the other essential parts of the cochlea, as the basilar membrane, the ciliated cells in connection with the terminations of the nerves, and Corti's membrane, which stands opposite the ends of these cilia, are all present. Hence it becomes very probable that Corti's arches play only a secondary part in the function of the cochlea. Perhaps we might look for the effect of Corti's arches in their power, as relatively firm objects, on transmitting the vibrations of the basilar membrane to small limited regions of the upper part of the relatively thick nervous fillet, better than it could be done by the immediate communication of the vibrations of the basilar membrane through the soft mass of this fillet. Close to the outside of the upper end of the arch, connected with it by the stiffer fibrils of the membrana reticularis, are the ciliated cells of the nervous fillet. In birds on the other hand, the ciliated cells form a thin stratum upon the basilar membrane, and this stratum can readily receive limited vibrations from the membrane, without communicating them too far sideways.

"According to this view Corti's arches, in the last resort, will be the means of transmitting the vibrations received from the basilar membrane to the terminal appendages of the conducting nerve. In this sense the reader is requested hereafter to understand references to the vibrations, proper tone, and intonation of Corti's arches; the intonation meant is that which they receive through their connection with the corresponding part of the basilar membrane.

"According to Waldeyer there are about 4500 outer arch fibres in the human cochlea. If we deduct 300 for the simple tones which lie beyond musical limits,

and cannot have their pitch perfectly apprehended, there remains 4200 for the seven octaves of musical instruments, that is, 600 for every Octave, 50 for every Semitone (that is, 1 for every 2 cents); certainly quite enough to explain the power of distinguishing small parts of a Semitone.

"To draw further conclusions from our hypothesis, when a simple tone is presented to the ear, those Corti's arches which are nearly or exactly in unison with it will be strongly excited, and the rest only slightly or not at all. Hence every simple tone of determinate pitch will be felt only by certain nerve fibres, and simple tones of different pitch will excite different fibres. When a compound musical tone or chord is presented to the ear, all those elastic bodies will be excited, which have a proper pitch corresponding to the various individual tones contained in the whole mass of tones, and hence by properly directing all attention, all individual sensations of the individual simpler tones can be perceived. The cord must be resolved into its individual compound tones, and the compound tone into its individual harmonic partial tones.

"This also explains how it is that the ear resolves motion of the air into pendular vibrations and no other. Any particle of air can, of course, execute only one motion at a time. That we considered such a motion mathematically as a sum of pendular vibrations, was in the first instance merely an arbitrary assumption to facilitate theory, and had no meaning in nature. The first meaning in nature that we found for this resolution came from considering sympathetic vibration, when we discovered that a motion which was not pendular could produce sympathetic vibrations in bodies of those different pitches, which corresponded to the harmonic upper partial tones. And now our hypothesis has also reduced the phenomenon of hearing to that of sympathetic vibration, and thus furnishes a reason why an originally simple periodic vibration of the air produces a sum of different sensations, and hence also appears as compound to our perceptions.

"The sensation of different pitch would consequently be a sensation in different nerve fibres. The sensation of a quality of tone would depend upon the power of a given compound tone to set in vibration not only those of Corti's arches which correspond to its prime tone but also a series of other arches and hence to excite sensation in several different groups of nerve fibres.

"Physiologically it should be observed that the present assumption produces sensations which differ qualitatively according to pitch and quality of tone, to a difference in the nerve fibres which are excited. This is a step similar to that taken in a wider field by Johannes Miller in his theory of the specific energies of sense. He has shown that the difference in the sensations due to various senses, does not depend upon the actions which excite them, but upon the various nervous arrangements which receive them. We can convince ourselves experimentally that in whatever manner the optic nerve and its expansion, the retina of the eye, may be excited, by light, by twitching, by pressure, or by electricity, the result is never anthing but a sensation of light, and that the tactual nerves, on the contrary, never give out sensations of light or of hearing or of taste. The same solar rays which are felt as light by the eye are felt by the nerves of the hand as heat; the same agitations which are felt by the hand as twitterings, are tones to the ear."

When Helmholtz formulated this resonance theory, as already stated, the anatomy of the cochlea was not fully understood. From the standpoint of physics it was a beautiful explanation; but with the

further observations of the anatomists already mentioned and those of others such at Böttcher,[8] Nuel[78] and Retzius,[84] who found that many of the structures of the cochlea were different from what Helmholtz had supposed, it was then that other explanations were sought. Helmholtz had apparently disregarded the findings of Reissner[83] and Kölliker[65] that the scala vestibuli and scala tympani were separated by another space, the scala media, and that incoming wave movements would have to pass through Reissner's membrane and the content of the cochlear duct before they reached the basilar membrane.

It was hard to understand how the specific parts of the basilar membrane could be selectively responsive to specific wave frequencies since the whole cochlear duct complex with its contained fluid must produce extreme dampening. The finding that the basilar membrane is not a simple membrane with only radial fibers but that in many animals, especially birds, it is quite thick with fibers running in all directions, still further detracted from the theory. Other objections were that the rods of Corti are only supporting elements and are not connected with nerves, and that such structures as Reissner's membrane and the tectorial membrane and the heavy cells of Claudius which overlie the basilar membrane were not considered in the theory. In 1898 Rutherford[86] went so far as to say, "I submit, therefore, that the minute structure of the cochlea places insuperable difficulties in the way of Helmholtz's and Hensen's theory, and I cannot but think that if Helmholtz had known the structure of the cochlea in mammals, but more especially in birds as described by Retzius, the resonance theory would never had been proposed."

As early as 1865 Rinne is quoted by various authors as considering the entire basilar membrane to vibrate to every tone. Wrightson and Keith[104] state that Wrightson in 1876 first presented his view that the basilar membrane vibrated as a whole. Voltalini, according to Knudsen and Jones,[67] or Volliolini in 1885, according to Wrightson and Keith,[104] and Voltolini, according to Hardesty,[42] also supported the idea that the entire basilar membrane is set into motion by every tone.

The first definite attempt to substitute a new theory of hearing for that of Helmholtz came in 1886 when William Rutherford,[85] at the meeting of the British Association held in Birmingham, gave a popular evening lecture in which he presented for the first time a hypothesis which could be called the "telephone theory" of the sense of hearing.

The theory states that the cochlea does not act on the principle of sympathetic vibration, but that the hairs of all its auditory cells

vibrate to every tone just as the drum of the ear does; that there is no analysis of complex vibrations in the cochlea or elsewhere in the peripheral mechanism of the ear; that the hair cells transform sound vibrations into nerve vibrations similar in frequency and amplitude to the sound vibrations; that simple and complex vibrations of nerve energy arrive in the sensory cells of the brain and there produce, not sound again of course, but the sensations of sound, the nature of which depends not upon the stimulation of different sensory cells, but on the frequency, amplitude and form of the vibrations coming into the cells, probably through all the fibers of the auditory nerve.

One of the arguments in favor of this theory was that he could explain differential tones or subjective tones which were difficult to explain by the Helmholtz theory.

L. Hermann[52] apparently was impressed by this argument of Rutherford and in 1891 published a paper "Zur Theorie der Combinationstöne" in which he presents the results of his experiments on the production of differential tones.

Rutherford[86] in his 1898 article on "Tone Sensation with Reference to the Function of the Cochlea" comments as follows on the work of Hermann:

"When two discordant tones are simultaneously produced, a beat is heard and if the vibrational difference between the two primary tones be sufficiently great, the successive beats give rise to the sensation of a third tone whose pitch is the vibrational difference between the two primary tones; for example, if the primary tones have respectively 880 and 1056 simple vibrations—namely, the notes A^1 and C^2—the pitch of the beat tone is 176 simple vibrations—namely, the note F.

"Hermann produced the two primary tones from two tuning forks, with a third fork at rest, but capable of resonating to the beat note if it had been produced objectively, but the third fork remained unaffected, although the observer heard the beat tone loudly. He, therefore, concluded that if the beat tone failed to excite a resonator outside the ear it could not affect any supposed resonator in the cochlea; consequently it must be a purely subjective phenomenon arising from the conflict of vibrations in the auditory center. That experiment, which has been fully confirmed, proves that the auditory nerve transmits to the sensorium vibrations of the same frequency as the sound waves and that they produce in the auditory center harmony or discord, according to their relative numbers. Hermann's conclusion from these experiments is that 'there is no alternative but to drop the Helmholtz hypothesis of resonators in the ear, although so elegant.' "

Hermann in later years seems to have returned to Helmholtz's theory. In his *Lehrbuch der Physiology*[53] published in 1896 he discusses several of the problems which are difficult to explain by the resonance theory. One of these is that the supposed strings in the basilar membrane are so short that it seems impossible that they should respond to incoming vibrations. He (Hermann) writes:

"It remains unbelievable that such short strings (.5 to .05 mm.) should have such low tonal responses as occur in music. If one considers the weighting down by endolymph the tension would have to be so exceedingly small that it seems impossible that they could resonate to such low tones. This difficulty can be eliminated if one considers the resonators not as elastic but as nervous structures of specific peculiarities."

It will be remembered that in his earlier articles he stated that the subjective tone or beat tone could not vibrate a resonator and that therefore the Helmholtz hypothesis of resonators was untenable. But in 1896 he writes:

"Another difficulty is the perception of the third tone obtained when two tones are combined. The perception of periods, which cannot stimulate resonators, can be explained by the hypothesis, that between the resonators and the acoustic fiber, cells are wedged which through the peridocity of the resonators are stimulated."

WALLER'S PRESSURE-PATTERN THEORY

In 1891 Waller[94] presented his pressure-pattern theory which is similar to that of Rutherford except in one respect. Rutherford claimed the stiff hair of the hair cells was set in motion by the tectorial membrane and thus the stimulation was transmitted to the nerves, while Waller considered the basilar membrane as the receptive structure. Here is Waller's theory in his own words:

"This limited title has been substituted for the more comprehensive title 'a new theory of auditory excitation' in consequence of the fact that Rutherford has offered a theory of very similar proport to that which I am about to sketch. The title as it stands denotes, however, what I regard as a main point of hypothesis, which in fact was the point starting from which I came to a position similar to that of Rutherford. Regarding the membrana basilaris as a narrow sickleshaped membrane (about 25 mm. long increasing from a breadth of about 1/20 mm. at the base to about ½ mm. at the apex of the cochlea) which carries upon its upper surface a field of hair cells overlaid by the membrana tectoria, the idea suggests itself that this membrane is an internal membrana tympani repeating in miniature the minute vibrations of the external membrana tympani and stapes, thus producing auditory excitations by mode of what may be termed 'pressure-patterns' of the hair surface against the membrana tectoria.

"This was the starting point, and in casting about for an analogy, a telephone membrane at once presented itself as the most familiar object. This telephone comparison has in fact already been made by Rutherford and contrasted with what may be shortly referred to as the piano theory of Helmholtz. Whereas on the *latter theory*, compound vibrations are analyzed and their components picked up by individual radial fibers of the basilar membrane, on the *former theory* compound vibrations are not analyzed at the periphery but reported 'telsquels' by the entire membrane.

"Examining the weight of evidence brought up in support of the consonation theory, viz., the observations of V. Hensen on auditory organs of Crustacea, and

those of Baginsky on dogs with partial destruction of the cochlea, I think we must admit that the consideration of alternative theories is not excluded."

In 1890 another investigation was published by Ewald[27] which seemed to be adverse to the theory of Helmholtz. Ewald destroyed the cochlea of pigeons and they were still able to hear at some distance with only the cochlear nerve intact.

It seems that these reports of Hermann,[52] Waller[94] and Ewald[27] gave Rutherford new faith in his telephone theory. In 1898 after a silence of 12 years he published a paper[86] entitled, "Tone Sensation with Reference to the Function of the Cochlea." In this paper he makes the following statement:

"At the meeting of the British Association, . . . I gave a popular evening lecture on the Sense of Hearing,[85] in which I proposed a new theory of tone sensation in opposition to the well-known theory of Helmholtz. . . . Although my theory has been accepted by a few physiologists, the majority, . . . still adhere to the Helmholtz theory, although there are evident signs that the implicit faith so long prevailing is gradually growing weaker."

He then refers to the structures of the cochlea which he considers important for a functional interpretation. "All are agreed that the scala media is the terminal organ that enables us to appreciate musical sounds."

He describes the basilar membrane as a dense membrane, especially in birds where the fibers run in several directions which make it unfit as a selective resonator.

He places great emphasis on the hair and the hair cells as the terminal receptors. The following selected passages from his article will tell his story:

"Since Corti's hair cells are the true auditory terminals through which the nerve fibers are impressed, their structure is of special importance. . . . The hair cells do not reach the basilar membrane.

"About 20 short, stiff, rod-like hairs project from the free extremity of each cell, their inner ends passing a very short way into the cytoplasm. Nerve fibrils terminate around the inner ends of the hair cells.

"Since the sound waves reach the hair cells through Corti's membrane (tectorial membrane) the nature of that membrane and its relation to the hairs are, therefore, important. . . .

"Retzius[84] describes it as 'homogeneous and semi-gelatinous' and states that the auditory hairs either project into or are in contact with it. If that be their anatomical relation the membrane and hairs must vibrate as one mass, like the membrane and ossicles of the tympanum.

"When the sound wave in its phase of condensation arrives in the scala vestibuli it is transmitted through Reissner's membrane to the fluid in the cochlear canal, thence through Corti's membrane, Corti's organ, the basilar membrane, to

the fluid in the scala tympani and membrane of the fenestra rotunda. All these parts oscillate back again in the rarefaction phase of the wave. It is admitted that the wave arrives at Corti's organ throughout the whole length of the cochlear canal at practically the same moment. The short, stiff, auditory hairs probably transmit the vibrations of Corti's membrane to Corti's cells in the same manner that they are transmitted by the columella of the bird's tympanum from the membrani tympani to the membrane of the oval window."

He considers chemical stimulation but thinks it impossible. He then says:

"It, therefore, seems reasonable to dismiss the idea of any special chemical action in Corti's cells and to assume that their action is probably mechanical. . . . The sensory apparatus is stimulated mechanically by the alternations of pressure in sound waves. This view is supported by observations of Ewald[27] that after complete destruction of the labyrinth and tympanic apparatus on both sides of the head in the pigeon the animal still gives signs of hearing sounds made at a distance. . . ."

THE TELEPHONE THEORY

Rutherford[86] states his theory in the following words:

"Eighteen years ago (1880) while thinking over the great difficulties that beset the resonance theory, it occurred to me that the telephone might by analogy help us to better understand the action of the cochlea. The telephone transforms sound vibrations, however complex, into electrical currents of corresponding frequency, amplitude, and wave form, and these in turn are retransformed into sound vibrations similar to those received. The theory of hearing which I was led to propose is that there may be no analysis of sound in the cochlea; and that all the hair cells may be affected by every sound, simple or complex, and through them the sound waves transmitted into nerve vibrations of corresponding frequency, amplitude, and wave form; that in the sensorium the nerve vibrations give rise to sensations varying in quality with that of the incoming impulses. My theory has been termed the telephone theory of hearing, and no doubt it is a convenient term to distinguish it from the resonance theory, but I wish it to be understood that I never regarded the hair cells as transforming the mechanical vibrations of sound into an entirely different mode of motion analogous to electricity. The essential point in my theory is simply this, that there is no analysis of sound waves in the cochlea. Theoretically a single hair cell, nerve fiber and sensory cell should suffice to give us all the different sound sensations; but probably the greatest number of auditory terminals and sensory cells renders us far better able to feel slight differences in the quality of sounds. My theory of tone sensation has the advantage of carrying the physical cause of harmony into the auditory center, and the validity of the argument I advanced in 1886 was afterwards proved by Hermann by experiments on the production of differential tones.

"Although the telephone theory of the action of the cochlea enables us to understand the production of sensations of harmony and discord, it still leaves much that is in the highest degree obscure."

We have already discussed Waller's pressure-pattern theory and that the main difference between it and the Rutherford theory is that Waller considers the basilar membrane as the structure that imparts

the impulse to the hair cells. Rutherford makes the following statement regarding it:

"I own that I have had great difficulty in seeing how the action of the cochlea is to be explained by Dr. Waller's theory. If the hairs of Corti's cells are to be subjected to different pressures in different parts of the cochlea, dependent on the unequal vibration of a tense membrane, it seems to me that such membrane ought to be undamped and placed in contact with the hairs of Corti's cells on the side of the entering sound. But the basilar membrane is heavily damped and is reached by the sound wave only after having traversed Corti's cells. I have already used this argument against Henson's theory and it seems to me to be no less applicable to that under consideration. But all our theories are open to criticism on a subject so obscure; none of us can say that we have a theory capable of explaining all the facts which have been ascertained regarding the cochlea, and this will become very evident when I have alluded to the results of experiments on animals and pathological observations on man."

Rutherford certainly was open-minded and was always ready to accept experimental and pathological findings. He cites experiments which support his theory but also those which support the theory of Helmholtz. He reported the work of Baginsky[5] on the dog and Corradi[15] on the guinea pig in which they destroyed the apical part of the cochlea with the result that the animals were responsive to high tones but not to low tones. Stepanow[90] also working on guinea pigs, obtained results directly opposed to the above findings. In a large series of guinea pigs he removed the apical turns of the cochlea and yet the animals responded to both high and low tones. Stepanow[91] also observed the case of a young man who had received a severe blow on his left ear which eventually led to exfoliation of the apical portion of the cochlea to the extent of one and a half turns of the tube. Hearing tests were made in the presence of a physicist and a physiologist and all were satisfied that the man could hear low as well as high tones. Gruber[34] reported a case of a boy, aged 14 years, from whose meatus he removed sequestrum consisting of the two apical turns. He stated that the pitch of the notes made no difference and that the boy could correctly imitate the note. In a case reported by Moos and Steinbrügge[77] there was extensive nerve atrophy to the basal turn associated with the loss of the audition of high tones. Regarding this case Rutherford writes as follows:

"The books, in referring to this case, assume that the nerve atrophy in the basal turn was the cause of the deafness to high tones; but singularly enough the two eminent aurists whom I have quoted regarded the nerve atrophy as possibly the result of inactivity owing to nontransmission of vibrations of high tones through the labyrinth because of the high pressure of the lymph which they were satisfied had been of long standing. In support of this hypothesis they refer to experiments made by Bernett under the direction of Helmholtz who found that high artificial pressure of fluid in the labyrinth diminishes the transmission of vibrations of low

but more especially of high tones to the round fenestra, and that when the pressure is greatly increased the vibrations of high tones are abolished."

Two cases were reported by Bezold.[7] Only one of these is sufficiently accurate to be cited. A woman of 65 years had a loss of tone perception at both ends of the audible scale. The patient died three weeks after examination. Upon examination of the ears he found complete ankylosis of the stapes which he considered the reason for the loss of low tone perception. Examination of the cochlea showed the absence of nerve fibers in the spiral lamina near the round window and moderate increasing atrophy in the rest of the first turn. There was a trace of atrophy in the second turn. The rest of the nerve fibers stained well with Weigert's method and Corti's organ was well preserved except in the first turn.

Rutherford referred to these cases to show that there is support for both theories and concludes that both theories are open to criticism.

EWALD'S SOUND-PATTERN THEORY

During the decade following the presentation of the telephone theory by Rutherford there appeared an epidemic of theories of hearing. Some of these favored the Helmholtz theory but either tried to modify it or deviated from it; others gave various explanations that were in line with the telephone theory and one tried to give a theory that was on middle ground and embodied the principles of both the resonance and telephone theories. All tried to explain away the objections raised against the existing hypotheses.

In 1899 Ewald[28] published his "sound picture" or "sound pattern" theory. He had experimented with loosely stretched rubber membranes which he set in motion by means of tuning forks. When a certain tuning fork is sounded, the rubber membrane will vibrate and one can observe on this membrane a certain resulting pattern of standing waves. If another tuning fork is sounded of a different frequency, a different pattern of standing waves is formed.

Ewald considered that in the ear the basilar membrane can pick up vibrations in the same way as does the rubber membrane. He felt that the portion of the basilar membrane between the pillars of Corti was especially adapted to do this, since it was thin and not loaded with large cells as is the rest of the basilar membrane. This sound picture is then transported somehow, probably through the organ of Corti, to the nerve fibers. He, like Rutherford, considered that all nerve fibers in the ear are generally alike and any fiber can transmit any vibrations. The sound picture on the basilar membrane is then

transferred to the brain in toto. Damage to an individual fiber here and there thus would make little or no difference in the total picture, whereas in the resonance theory damage to a fiber would mean loss of the specific pitch transmitted by such a fiber. Here is Ewald's sound pattern theory in his own words:

> "In the ear, by means of tone vibrations, a wave picture is produced on the basilar membrane whose duty it is to present this special picture as a link in the chain of the transportation mechanism between sound and sound perception. This is the principle of the "Sound Pattern" theory. No more and no less."

In support of his theory he says: "Most investigators who have carefully studied the theories of hearing conclude that the Resonance Theory is not sufficient. . . . Hermann,[52] Mach[73] and König[66] have showed this best."

EMILE TER KUILE'S THEORY

In 1900 ter Kuile[63, 64] presented his theory in two publications. In the first and part of the second he discusses at length the mechanism for stimulating the hair cells, which he says must be considered the principle element to which the energy from the basilar membrane must be transferred.

He writes: "There exists no clear statement as to how the energy from the basilar membrane is transferred to the hair cells."

The usual concept was that the hairs bump against the tectorial membrane when the basilar membrane moves. Thus Helmholtz writes: ". . . these hair cells which one finds everywhere and whose hair are so placed that they can bump against the Corti's membrane with the movement of the basilar membrane."

Hensen also had presented this idea. He also stated that this bumping was possible because the tectorial membrane was stationary.

Emile ter Kuile offers five objections to the resonance theory:

1. The tectorial membrane is not stationary. All the structures in the cochlear duct, including Reissner's membrane and the tectorial membrane, must move with the pressure changes which affect the basilar membrane.

2. The hair lies in a position where there is very little up-and-down movement.

3. In birds and reptiles there are many hair cells which lie in a position where they cannot move with the basilar membrane.

4. No special function is known for the membrana reticularis and the pillar cells are absent in birds and reptiles.

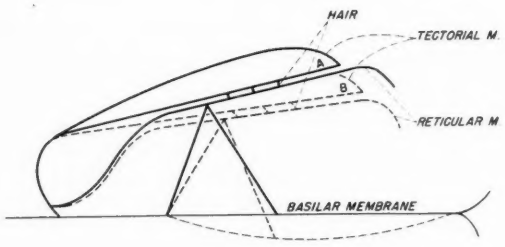

Fig. 2.—Emile ter Kuile's idea of the movement of the hair of the hair cells with the movement of the basilar membrane.

5. He has a theory to offer which does not have the above-mentioned faults.

He places great emphasis on the tectorial membrane in relation to the hair cells. He says that the tectorial membrane must be important for it is present in the earliest appearance of the basilar membrane in anura. This membrane always overlies the hair cells, even in reptiles and birds where hair cells are scattered throughout the epithelium. Again the hair always lies between two plane surfaces, the underside of the tectorial membrane and the surface of the cells of the organ of Corti. He writes: "When I see two plane surfaces with hair between them and tightened to both, the thought forces itself on me that these surfaces must glide back and forth and thus cause the hair to move back and forth."

He then proceeds to describe the mechanism in the cochlea by means of which this type of movement of the hair cells can be effected. He calls attention to the fact that the inner pillar cell is anchored on the tip of the bony spiral lamina and as such there can be no appreciable movement of the base of this inner pillar cell when the basilar membrane moves. The outer pillar cell, however, is anchored on the basilar membrane and could move up and down with the up-and-down movement of the basilar membrane. Since the heads of the pillar cells meet and the foot of only the outer one is so attached to the basilar membrane that it alone can move up and down, the movement of the entire organ of Corti complex must therefore be a type of hinge movement with the foot of the inner pillar cell acting as the hinge. The resultant effect of this movement on the hairs of the hair cell is a back-and-forth movement as can be seen from his diagram (Fig. 2).

Regarding Ewald's theory, ter Kuile speaks kindly but says that, if Ewald had considered the entire basilar membrane in the recording of the standing wave instead of only that portion between the two pillar cells, then his theory could have been applied to ter Kuile's theory of the side movement of the hair.

In introducing his own theory ter Kuile[64] states that:

"It is not clear whether Helmholtz considered mass movement or molecular vibration as the cause for the stimulation of the specific part of the cochlea. Nor does he make clear whether he considers these specific structures as *resonators* or only tone *analyzers* of a special type such as physical or as physiological resonators.

"Since he employed the physical laws of sympathetic vibration, *resonance* and *dampening* it appears that he must have considered them as physical resonators. In that case it would be unnecessary to transform the molecular movement of sound into mass movements.

"Most authors feel that Helmholtz thought that the resonators were stimulated by molecular motion."

On this basis Bonnier[10] criticized Helmholtz's theory. Bonnier says, "It is impossible for any of parts of the cochlea to resonate such low tones as we hear and that therefore stimulation can only result through mass movement."

Ter Kuile thinks that Helmholtz actually thought in terms of mass movement and quotes Helmholtz as saying that the only reason why the radial fibers are bound together by cross fibers "is to furnish a handle on which the pressure of the fluid can act to stimulate the strings." Were it only a matter of physical stimulation of strings by molecular action, then such string would need no handle.

Emile ter Kuile states his theory in these words:

"The theory of hearing which I give is a result of the application of Ed. Weber's principle of mass movement.

"The mechanical function of the middle ear apparatus is to transform the molecular movement of the air into mass movement of the labyrinthine fluid. Within the ear there can be no sound. The tympanic membrane is the boundary between sound and not sound. The ear does not serve to resonate the sound but to kill it; that is to transform it into energy of some other sort, namely, nerve energy.

"When the stapes is pressed inward a quantity of lymph in the scala vestibuli will be moved toward the fenestra rotunda, because this is the only movable place. If there were no basilar membrane this lymph movement would be as direct as possible and occur only in the proximal part. There exists, however, between the two windows a more or less displaceable membrane, the zona pectinata (between outer pillar and spiral ligament). The proximal end of this membrane is narrow and becomes wider more distally. Thus only a small displacement can take place at the proximal end and the movement moves along the membrane to a certain

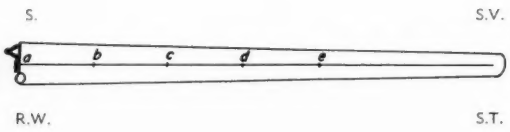

Fig. 3.—Emile ter Kuile's idea of the extent of movement of the basilar membrane. A high frequency tone would displace the membrane from *a* to a point *b*, a somewhat lower frequency from *a* to a point *c*, etc. All tones would displace the area *a* to *b* but lower tones would displace more of the membrane. *S*, stapes; *RW*, round window; *SV*, scala vestibuli; *ST*, scala tympani.

point where all displacement is complete. (That is when the stapes has reached its greatest inward movement.) As the stapes moves outward the reverse action takes place and the lymph becomes stationary. Now a low tone with large waves must travel to a point more distal on the basement membrane before displacement is complete than is the case for a high tone with small or short waves. Thus if a certain tone will travel along the basement membrane for a distance of N then a higher tone with 4 times the number of vibrations will travel only ¼ the distance of N."

According to this all waves stimulate the proximal part but the lower the tone the more distally will they stimulate the basilar membrane (Fig. 3). This movement of the basilar membrane is transferred to the hair of the hair cells. Thus for a high tone a few proximal cells have their hair stimulated very rapidly, whereas for a lower tone more cells are stimulated but at a slower rate. Thus at the proximal end all tones stimulate the hair cells but at different rates, but distally each tone has a specific extent.

He places emphasis on the specific extent to which a specific tone displaces the basilar membrane. This is determined largely by the time required for the stapes to make one inward movement. Amplitude is also concerned but to a lesser degree, since it more specifically determines the degree of movement of the basilar membrane. Of two different tones with the same amplitude, the one with the lower pitch will displace the basilar membrane to a lesser degree but over a greater extent than a tone of higher pitch. Thus, for a given tone the extent or displacement is always much the same. He states that within the ear there can be but little difference in amplitude for a given pitch. It may be, however, that with greatly increased amplitude the movement along the membrane may be speeded up a very little, so that it will terminate at a point slightly further along than for a lower intensity. This he thinks would explain the known fact

that a tone sounded loudly will be perceived as of a lower pitch than when the same frequency is not sounded so loudly.

This theory has been referred to by Wilkinson[100] as the "traveling bulge" theory.

Emile ter Kuile[64] takes a stand intermediate between that of Helmholtz and Rutherford as gathered from these words:

"Since there are many more hair cells than pillars there can be more critical analysis than is possible with the Helmholtz theory where the pillars are considered important analyzers.

"According to the *Helmholtz* theory the superposition of simple tones is analyzed into their components. According to Bonnier[101] and necessarily also according to Ewald there is a repetition of superposition curves. Bonnier does not believe in tone analysis. According to my [ter Kuile] theory an analysis occurs but also a superposition and the two do not interfere with each other; the physical superposition is changed to a physiological one, which is of a higher order, because the components are not lost."

MAX MEYER'S THEORY (THE TRAVELING BULGE THEORY)

Max Meyer, a contemporary of ter Kuile, presented a theory much like that of ter Kuile but differing from it in certain respects. Meyer's[74] first account appeared in 1898, two years prior to that of ter Kuile. The latter apparently did not know of the former's work and so the two workers reached their conclusions independently. Meyer published a series of articles but the one of 1900[75] in which he comments on the theory of ter Kuile, gives his views most clearly. Again in 1907 Meyer[76] published a very extended account of his concept on the mechanics of the inner ear.

Meyer agrees with ter Kuile that, with the initial movement of the stapes, the wall between the scala vestibuli and scala tympani is bent downward at its basilar end near the round window and, as the stapes moves further inward, the movement of the basilar membrane progresses toward the apex but only so long as the stapes moves inward. As soon as the stapes stops its inward movement, the movement along the basilar membrane stops. As the stapes moves outward, the basilar membrane again responds but this time it moves upward. This movement again starts at the basal end and moves toward the apex. This movement again proceeds only as long as the stapes moves. When the stapes halts, the movement on the membrane stops. Both men are agreed that for low pitches the movement along the basilar membrane is over a longer distance than for higher pitches. Both agree that at the basal end of the membrane all pitches are superimposed.

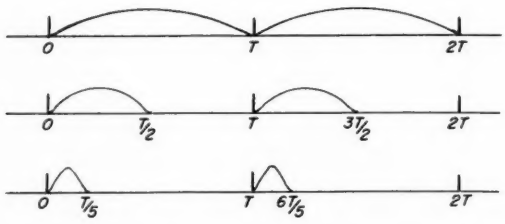

Fig. 4.—Meyer's illustration of how tones of different pitches, when only one wave of each is allowed to reach the ear in a given unit of time, will produce one and the same pitch depending on the length of the unit of time.

One of the chief differences is that ter Kuile thinks that the distance over which the basilar membrane is moved is determined by the time required by the stapes to make its movement in one direction, whereas according to Meyer the distance is determined largely by the amplitude of the waves. Thus according to ter Kuile the perception of pitch depends on the extent to which the basilar membrane is stimulated.

Meyer's chief criticism is that according to ter Kuile's theory a single wave could produce the sense of pitch. This he says is not possible but that at least two successive waves must be perceived to determine pitch. Thus according to him the number of times per unit of time that the membrane and hair cells are stimulated determines the pitch we detect and not the specific extent over which the membrane is moved. He cites the known fact that if a pitch is sounded which has 100 vibrations per second but every second wave is masked out or interrupted, then only 50 vibrations per second reach the ear. The pitch perceived then is that of 50 per second and not that of 100 even though the actual length of the wave occupies only one-half the natural period of the pitch perceived. According to ter Kuile the length of the individual wave should determine the pitch, while Meyer says pitch depends on the frequency of stimulation per unit of time irrespective of the length of the wave (Fig. 4).

Meyer does not lean toward the Helmholtz theory. He writes: "I ascribe to each cell the capability to sense each frequency as a specific quality."

THE MAXIMAL AMPLITUDE THEORY OF GRAY

The theories of this period were not all favorable to the telephone theory. Gray[33] in 1900 presented what is known as the theory of maximal amplitudes, which is a modification of the Helmholtz resonance theory. Gray made the observation that the spiral ligament was much more massive and stronger in the basilar end of the cochlea than in the apical end. This observation coupled with the observed facts that the basilar membrane gradually becomes wider and the tectorial membrane more massive in passing from the basal to the apical turns convinced Gray that these anatomic structures favored the belief of cochlear analysis of pitch. Here are Gray's own words:

"In the first place, it is clear that since the ligamentum spirale consists either of unstriped muscular fibre, or, more probably of fibrous connective tissue, it must produce tension on the basilar membrane. And further, since as above described, it increases greatly in size from the apex of the cochlea to the base, then the tension exerted by it on the basilar membrane must increase to a corresponding extent. Now if we look upon the basilar membrane as a series of strings, as Helmholtz did, then these strings must be under gradually increasing tension the further towards the base of the cochlea we go and this being so, their vibration frequency must increase correspondingly. The fact of the increasing size and strength of the ligamentum spirale downward, therefore, strengthens immensely the view that sound is analyzed into its simple harmonic constituents by the cochlea. The change in size of the structures of the organ of Corti and the diminution in breadth of the basilar membrane, might be mere coincidences or might not alone be sufficient grounds for supporting this view, but when we have associated with these the remaining factor, tension, which would affect the pitch of the membrane, then the evidence appears overwhelming.

"The fact of the increasing size of the ligamentum spirale downward, which has not claimed the attention of the physiologists, appears to the writer to be exceedingly strong evidence in favour of the view that sound is analyzed in the cochlea so far as it is ever analyzed at all."

Gray admits that there are several serious objections to the resonance theory. One of these is that differential, or so-called subjective, tones are not resonated and yet we hear them. Gray writes thus:

"These tones, according to those who put forward this objection, cannot set resonators in vibration, and must, therefore, be generated in the mind of the listener, which if it were true, would be fatal to the theory that sound is analyzed in the cochlea by sympathetic resonance. Recent investigations, however, by Forsyth and Sowter (Proc. Roy. Soc. London LXXXIII, 1896, P. 393) have shown that differential tones can be resonated if the resonator is sufficiently accurate. Furthermore, it has been pointed out that differential tones may be generated in the middle ear—Helmholtz (Tonempf. 3d Edit., trans. by Ellis, P. 237), Preyer (Wiedmann's Annal. XXXVIII, P. 131) and others; and this would fully account for the fact that it is admittedly difficult, though not impossible, to resonate differential tones."

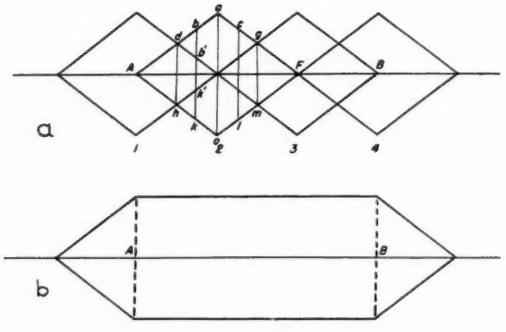

Fig. 5.—Illustrations from Gray somewhat modified. 1, 2, 3 and 4 indicate four distinct tones which overlap each other. The sum of the overlapping tones produce an amplitude equal to the maximum of the individual tones and thus a maximal stimulation occurs over the basilar membrane from A-B in *b*. In this span it is impossible to separate the individual tones and the result is a noise.

Gray accepted these meager evidences that differential tones might be resonated and calmly dismissed this serious objection to the Helmholtz theory.

According to the resonance theory each fiber in the basilar membrane would respond specifically to a specific tone. Helmholtz did admit that with a loudly sounded tone one or two fibers on either side of the specific fiber could vibrate in sympathy. Rutherford[86] had strongly challenged this interpretation and stated that the fibers in the membrane were so intimately bound together that an individual fiber could not vibrate alone.

Gray[33] agrees with Rutherford on this point, but proceeds to explain why this is not an objection to the resonance theory. When a pure tone calls forth a movement of the basilar membrane *not only* one fiber or one set of hair cells or one nerve fiber but quite a number are stimulated. Thus a pure tone may stimulate a segment of the basilar membrane say from *A* to *F* (Fig. 5). The maximum stimulation would occur at the segment *ao*, but the segments *bk*, *cl* and *gm* and *dh*, would be stimulated to a progressively lesser degree. The corresponding hair cells and nerve fibers would be stimulated. The main point in his theory, however, is that only that nerve which

received the maximal stimulation will transmit the impulse to the brain.

According to the Helmholtz theory noises could not be accounted for; Gray explains them in the following manner: If four slightly different tones stimulate the basilar membrane in a manner shown in Fig. 5 so that there is an overlapping of the stimulation on the membrane, then the sum of the stimulations would produce a continuous maximal stimulation of the membrane as shown in Fig. 5, b, from *A* to *B*. With such a continuous maximal stimulation it is impossible to separate specific tones and the result is a noise.

Gray[33] is very specifically concerned with the nature of the transformation of the movement of the basilar membrane into nerve impulses. He thinks the vibrations of the basilar membrane produce touch or pressure stimulations in the hair cells and this touch or pressure impulse is transmitted through the nerve to the brain. In explanation of this view, let me quote Gray's own words:

"Since the hair cells follow every movement of the basilar membrane, then, in their upward displacement, the hairs and even to some extent the cells themselves, will be pressed against the tectorial membrane which in the living condition, lies like a pad over them.

"When, therefore, the hair cells are raised against the tectorial membrane, the latter will resist their upward progress and press them down against the nerve terminations at their bases. Further, the greater the amplitude of movement of the basilar membrane, the greater will be the pressure of the hairs and hair cells against the tectorial membrane and the greater will be the stimulation of the nerve fiber at the base of the haircell. When, therefore, a pure tone calls forth a movement of the basilar membrane, a considerable number of nerve fibers will be stimulated, but the intensity of the stimulation will be greatest in that nerve termination which lies at the base of the hair cell opposite the point of maximum movement of the basilar membrane. In the same way the movements of the membrane called forth by compound tones and noises are transferred into pressures upon nerve termination. Now no more perfect means could be devised for this purpose than the tectorial membrane. It is not attached to the vibrating portion of the cochlea, and it lies over the hairs like a pad so that the more they are projected upwards against it, the more firmly does it press the hair cells down against the nerve termination.

"By this means, therefore, the variations in amplitude of movement of the basilar membrane become transformed into exactly corresponding variations of pressure upon the nerve terminations. These variations of pressure are then sent to the brain and there analyzed in exactly the same way as the pressure variations in the sense of touch are analyzed. In fact, the auditory nerve may be looked upon purely as a nerve of touch, but vastly more delicate than any of the other nerves of touch. This is to be expected, for the supply of nerve fibers to the organ of Corti is far richer than to any part of the skin of corresponding dimensions.

"The theory of maximal amplitudes, as it may be termed, appears to account for the known facts concerning the sense of hearing as completely as the theory of

Helmholtz; of which it is a modification; and the objections urged against the latter theory cannot be urged against it. Thus many will not admit that each fiber of the basilar membrane or each arch of Corti can move independently of those adjacent to it which the Helmholtz theory requires. This objection cannot be urged against the theory proposed in this paper.

"Again Helmholtz's theory does not explain the existence of noise, unless the latter be regarded as purely psychological; even if that were admitted it does not explain why we cannot, under any circumstances, analyze a noise into its constituent simple tones. The theory proposed in this paper is exactly the reverse in this respect, for supposing it to be correct, then a sound is a noise when we cannot analyze it into its simple constituents; and if we are able to analyze, whether entirely or only partially, then a musical element appears.

"In this theory of hearing we have seen a remarkable analogy between that sense and the sense of touch.

"The pathological facts concerning the loss of high tones with diseases of the lower whorl of the cochlea is equally explicable by either this theory or that of Helmholtz."

THE PLACE OF THE TECTORIAL MEMBRANE

It will be recalled from the foregoing account that Hensen[51] followed Claudius' idea that sound impulses entered the cochlea via the round window and stimulated the basilar membrane from the scala tympani. At about the same time Helmholtz carefully described the middle ear mechanism and propounded the plausible hypothesis that the impulses were transmitted to the scala vestibuli via the stapes through the oval window. This view has been the predominant view. Helmholtz recognized two possibilities as to how such sound waves could reach the basilar membrane, either by passing along the scala vestibuli to the helicotrema and then to the scala tympani and there stimulate the basilar membrane or else the waves would have to traverse Reissner's membrane, the fluid of the cochlear duct and the organ of Corti before it could reach the basilar membrane. The latter view has been most generally accepted. Now it seems very strange that if this be the course of the incoming wave movement a stimulation of the tectorial membrane was not considered important, especially since it was considered by all as coming in contact with the hair of the hair cells, which hair cells, most investigators believed, were the structures which transferred the mechanical impulse to the nerve mechanism. No doubt the old idea of Hensen that the tectorial membrane could not be moved had diverted all attention from it.

In 1891 Ayers,[4] presented the first suggestion that the tectorial membrane might be stimulated by the sound waves. He compared it with the cupola of the crista ampullaris and said it was much more

sensitive to changes in the endolymph than the basilar membrane could possibly be. It can transfer its vibration directly to the hair cells.

In the presentation of his telephone theory in 1898 Rutherford[86] had a vague notion that the tectorial membrane was important in the pick-up of the sound waves as may be gathered from his own words:

"Since the sound waves reach the hair cells through Corti's membrane (tectorial membrane) the nature of that membrane and its relation to the hairs are, therefore, important. . . .

"Retzius describes it as homogeneous and semigelatinous and states that the auditory hair either project into or are in contact with it. If that be their anatomical relation the membrane and hairs must vibrate as one mass, like the membrane and ossicles of the tympanum."

In 1897 Siebenmann,[89] on page 314 of Bardeleben's *Handbuch der Anatomie*, indicates that he believes the tectorial membrane is an efficient structure for the reception of the sound waves and for the transmission of them to the hair cells. He states that the tectorial membrane lies nearest to the incoming sound waves and that of all the structures in the cochlea, it should be set into vibration the most easily and that its movements can be directly transferred to the hair of the underlying hair cells which in turn stimulate the acoustic nerve.

KISHI'S THEORY

In 1907 Kishi,[62] after citing anatomical objections to the basilar membrane as a vibrating mechanism of the resonance theory or any other theory, substitutes the tectorial membrane. He assumes that the tectorial membrane is attached at the inner margin (as all are agreed) but also at the outer end to the cells of Hensen. Thus the membrane would be a stretched membrane. He says that the tectorial membrane is markedly elastic in a radial direction. He assumes that radial fibers occur within the tectorial membrane and that the length of these fibers vary with the width of the tectorial membrane. Although he admits that he could not make accurate measurements of the length of these fibers because of distortion, he did conclude from his measurements that the fibers are three times as long in the apical region as in the basal region. He assumes that these fibers have different spans (vibrating lengths) with the longest at the apex and the shortest at the base. He believes these fibers capable of sympathetic vibration to all sound waves. Since the tectorial membrane is in contact with the hair of the hair cells, the resonating fibers of the membrane can transmit their vibrations to such hair cells.

SHAMBAUGH'S THEORY

In August, 1907, Shambaugh,[87] working independently, reached a conclusion similar to that of Kishi although differing in detail. He presents the anatomical objections to the basilar membrane as a resonating mechanism. His own observations of cases with the absence of a membranous basilar membrane add to his conclusion. After raising the various objections to the resonance theory as then known, he still states:

> "The fundamental principles in the resonator theory of Helmholtz still remain as the most plausible explanation of the various phenomena of tone perception. With the physiological and clinical facts which we possess, we can hardly escape accepting a resonator theory in some form."

Shambaugh selected the tectorial membrane as the organ which analyzed the incoming sound waves. In support of his view, he cites a number of anatomical characteristics of the tectorial membrane.

1. The tectorial membrane does not float free above the organ of Corti but is attached to the inner supporting cells by its stripe of Hensen and also to the hair of the hair cells. Retzius was one of the first to insist that the hair were attached to the tectorial membrane and this view was accepted and emphasized by Rutherford, ter Kuile and Kishi. Kishi and Shambaugh claimed that besides attachment to the hair the tectorial membrane was also attached to the organ of Corti at some other point.

2. The tectorial membrane is made up of a delicate semifluid substance in which occur delicate lamellae. To quote:

> "An immense number of delicate lamellae are found taking their origin from the portion of the membrane which rests on the labium vestibulare. The lamellae are more compact where they converge along the dorsum of the membrane, curve gracefully outward and downward toward the lower border of the membrane. These lamellae give the membrana tectoria somewhat the appearance of a soft feather. They vary in length with the varying size of the tectorial membrane. . . . They are supported and held together by an apparently homogeneous semifluid substance. The specific gravity of this substance appears to be the same as that of the endolymph in which the membrane is suspended."

He also describes and pictures the size and volume of the tectorial membrane in the different turns of the cochlea to show that it increases in size as it passes from the lower turn to the apical turn. He says:

> "It is seen at a glance that the size of the membrana tectoria near the apex of the cochlea is many hundred times its size near the beginning of the basal coil."

From these findings he makes the following deductions:

"The membrana tectoria is shown to be so constituted anatomically as to be capable of responding to the most delicate impulses passing through the endolymph. Furthermore, the great variation in size of this membrane from one end of the cochlea to the other, together with its lamellar structure, suggest the probable physical basis which renders it capable of acting the part of a resonator by responding in one part to impulses of a certain pitch and in another part to impulses of another pitch. . . . The vibrations in a part of the membrana tectoria produced by a particular tone must necessarily involve a considerable area of this structure. As a result a more or less extensive group of hair cells is stimulated. The nerve impulses arising from the stimulation of the several hair cells included in this group come together in the brain center of the cortex, where the tone picture forms the final step in the perception of this particular tone. When a tone slightly higher or lower than this one is produced, the same group of hair cells is stimulated, excepting for the addition of a few more cells at one end and the loss of a few cells at the other end of the area involved. The sum total of the impulses which reach the center in the brain is, therefore, different for every tone, however near they may be in the scale."

This description seems to fit into the picture of the place theory rather than the resonance theory. He claims that his theory accounts for such clinical or pathological conditions as tonal islands, diplacusis binauralis dysharmonica, and tinnitus aurium. He feels that all types of tinnitus can be traced to an intralabyrinthine pressure. Such pressure, he feels, disturbs the normal relationship of specific gravity between the tectorial membrane and the endolymph and thus causes stimulation of the hair cells.

HARDESTY'S CONCEPT OF THE TECTORIAL MEMBRANE

In 1908 Hardesty[42] in a lengthy account of his findings from very careful anatomical studies on the tectorial membrane concluded that this membrane is set into vibration by sound waves rather than the basilar membrane which he considers as "nothing more than a flat tendon." He differs in several respects from the conclusions of Shambaugh. "In the first place," he writes, "it is considered probable that the tectorial membrane does lie free over the organ of Corti and that the auditory hair do not project into it."

In the second place, he says, "It is not a lamellated structure." He describes it as follows:

"Its structure consists of multitudes of delicate fibers of unequal length, embedded into a transparent matrix of a soft, collagenous, semisolid character with marked adhesiveness."

He states that none of its fibers extend the entire width of the membrane, none are attached at both ends, and the greater number of them are attached at neither of their ends. Because of this he

thinks it "very improbable that the membrane is capable of acting as a sympathetic resonator."

He thinks the sound waves run along the length of the tectorial membrane but admits that the high frequencies may travel only along the membrane in the basilar turn since they may be sooner checked by the increasing mass of the tectorial membrane than lower frequencies. Thus he admits that there may be some analysis of pitches in the cochlea due to the anatomical structure of the membrane.

He states that he does not propose to elaborate another theory of hearing but says: "Leave is asked to merely suggest a modification of a theory already advanced, namely an application to the tectorial membrane of the telephone theory, heretofore applied exclusively to the basilar membrane."

In 1915 Hardesty[43] presented a paper setting forth the results of experiments with a model of the cochlea which he constructed. The results agreed with the interpretation he presented in his earlier paper that low tones could set the entire tectorial membrane in motion but high tones vibrated only the basal portion of the membrane. The extent to which the tectorial membrane is set into motion depends more on the pitch than on the amplitude. This interpretation of the action of the tectorial membrane in response to incoming sound waves is very similar to that which ter Kuile thought occurred in the basilar membrane.

During the decade following the attempted rise to fame of the tectorial membrane as the active transmitter of sound vibrations, numerous minor attempts were made at new theories of hearing, but none of these seem to contribute much. Lehmann,[68] in 1910, attempted to unite some of the merits of the Helmholtz and Ewald theories, while Watt,[96] in 1917, followed closely the theory of ter Kuile with minor modifications.

THE DISPLACEMENT THEORY OF WRIGHTSON AND KEITH

Wrightson and Keith[104] state that Wrightson as early as 1876 suggested that the basilar membrane vibrates as a whole, that a separate nerve stimulus is produced at every "crest", "trough" and "crossing point" of any sound wave, however complex that sound may be and that the brain will receive sufficient data to make possible a complete analysis of that sound. He suggested that each complete sound vibration is made up of four phases and that each phase acting against an elastic resistance can give rise to a displacement stimulus. He thought that each such displacement (four for each sound wave) re-

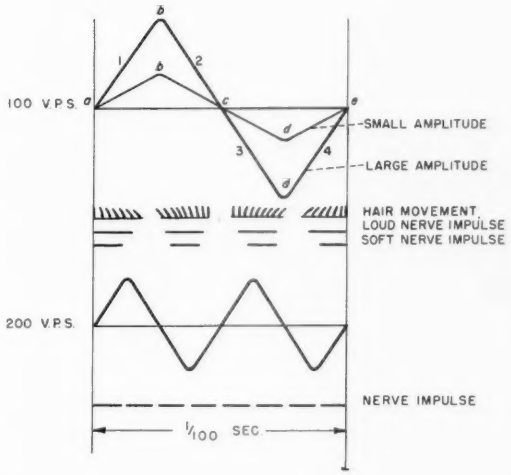

Fig. 6.—This figure illustrates Wrightson's and Keith's explanation of the four phases of a wave motion and their effect on the hair cells and on nerve impulses. It will be noted that for 100 v.p.s. the nerve is stimulated four times per unit of time while with 200 v.p.s. eight such stimulations occur in the same period of time. Pitch thus is determined by the number of stimulations per unit of time.

sulted in a rubbing or friction motion on the hair of the hair cells against the tectorial membrane, which irritation was transformed into a nerve stimulus.

Keith, an anatomist, aided Wrightson in analyzing the structure of the cochlea and as a result Wrightson and Keith together published a modification of Wrightson's theory in 1918. Their explanation of the stimulation of the hair of the hair cells is one of back-and-forth movement. Keith followed the interpretation of ter Kuile that the hair were in contact with the tectorial membrane and that the hinge movement of the organ of Corti caused a bending back and forth of the hair between the organ of Corti and the tectorial membrane. Ter Kuile thought there was one such bending for each wave but Wrightson and Keith state that these hair are bent in a different direction at four different intervals for each complete vibration. In Fig. 6 the four phases are represented. The segment *a-b* represents the in-

ward movement of the stapes, *b-c* the reverse movement to rest, *c-d* the outward movement of the stapes and *d-e* the reverse movement of the stapes to rest. The effect on the hair of the hair cells to each phase is as follows:

The hair at rest are perpendicular to the under surface of the tectorial membrane. During phase *a-b* the basilar membrane is pressed toward the scala tympani and the hair tips are bent inward; during phase *b-c* the hair are bent back to the point of rest; during *c-d* they are bent outward; and during *d-e* they are bent back to the position of rest. There is a momentary period of rest between each of these directions of bending. During each period of bending the nerve is stimulated. There is a brief period of rest before the next period of bending stimulates the nerve again. Thus for each wave motion the nerve is stimulated four times. Pitch is determined by the frequency with which such stimulations occur. They represent each stimulation by a dash and for each wave motion the four dashes are separated by a space for the moment of nonstimulation. They write.

"The length of the dashes represents the intensity or loudness; the number of spaces or intervals between the dashes stand for the pitch. The brain is supplied just as a telegraph operator is with all the data necessary to unravel the constituent elements of a complete message."

It is worthy of note that during the period following the presentation of the telephone theory by Rutherford[85] in 1886 very few attempts were made to strengthen the Helmholtz resonance theory. A few of the men mentioned suggested cochlear analysis of sounds but more in the sense of the place theory than the resonance theory. Thus ter Kuile[64] assumed cochlear localization but also superposition of pitches. Gray's maximal amplitude theory most nearly agrees with that of Helmholtz but he really is an exponent of the place theory since his method of nerve stimulation is compared to that of touch. Kishi[62] and Shambaugh[87] disagree with Helmholtz as to the function of the basilar membrane but accept the tectorial membrane as the resonating structure. The other men of this period tend toward the telephone theory to the extent that there is no specific analysis of tones in the cochlea.

Starting with 1920 there seems to be a distinct swing back toward the Helmholtz theory. Hartridge[44-46] and Wilkinson[100, 101] assumed that the basilar membrane is the active vibrating mechanism. They presented results based on calculations and physical experiments which seem to show that the actual fibers of the basilar membrane can be regarded as possessing the necessary requisites of length, tension and mass to satisfy the harp theory.

Hartridge,[46] knowing that the length and mass of the fibers in the basilar membrane was fairly well established, realized that the burden of proof rested upon the matter of tension. He cites instances of tension in animal tissues, such as hair and tendon, which were considerably greater than was demanded for fibers by the harp theory. Length, mass and tension of basilar fibers thus seem to be adequate for resonation of sounds.

He thinks that the transmission of the vibrations to the hair cells and the nerve is not by bending or pressure but by tension or traction. He writes:

> "When, therefore, the basilar membrane moves upward the tectorius would be urged upward too, but when the basilar membrane reaches the end of its motion and returns, the tectorius, possessing mass and therefore momentum, would tend to go on rising and would therefore pull on the processes of the hair-cells with the force needed to give to the tectorius that acceleration which will cause it to follow the down motion of the basilar membrane. According to this view the stimulus to the hair cells is not by pressure [Helmholtz] nor by friction [Wrightson], nor by bending [Keith] but by traction [Hartridge]."

Hartridge[45] cites another interesting experiment in which, by means of a siren, he could suddenly reverse phase in sound vibrations passing from holes in the siren. Such a reversal of phase produces a momentary cessation, and then a building up of the same tone sensation to its original loudness. Those telephone theories which assume that the action of the cochlea is "piston-like", that is, that the membrane within the cochlea follows the motion of the stapes in a "dead beat" fashion, would find it difficult to explain this phenomenon.

Wilkinson[100] thinks that the mass of the fibers of the basilar membrane is augmented by the cochlear fluid. He calls this "loading." He argues that those high frequencies affecting the basal part of the cochlea have a ready exit at the round window, so that only a small part of cochlear fluid is involved. Low frequency vibrations affecting the structures in the apex have to displace the whole column of cochlear fluid. This fluid constitutes a main factor in mass. At the base of the cochlea this factor of fluid loading is least, while at the apex it is most. Thus he thinks that, considering the length, tension and loading or mass of the fibers in the basilar membrane, all requirements for the harp theory are fulfilled.

THE SPACE-TIME PATTERN THEORY OF FLETCHER

In 1930 Fletcher[32] suggested a combination of two general theories of hearing. In the one it is assumed, so writes Fletcher, "that the time pattern of the wave motion in the air is transferred into a

space pattern in the inner ear so that the nerve impulses reaching the brain give us information concerning the time pattern of the wave motion by means of the location of the nerves which are stimulated." This he designates as the "space pattern" theory. Regarding the other or "time pattern" theory he says, "It is assumed that the time sequences are transmitted directly to the brain."

Fletcher in summarizing this space-time pattern theory makes the following statements:

"The pitch of a tone is determined both by the position of its maximum stimulation on the basilar membrane and also by the time pattern sent to the brain. The former is probably more important for the high tones and the latter for the low tones. The loudness is dependent upon the number of nerve impulses per second reaching the brain and possibly somewhat upon the extent of the stimulated patch. The experience called by psychologists 'volume' or 'extension' is no doubt identified with the length of the stimulated patch on the basilar membrane. This extension is carried to the brain and forms a portion of excited brain matter of a definite size. It is then this size that determines our sensation of the volume of a tone. Thus low pitched or complex tones have a large 'volume' while the high pitched tones have a small one.

"The time pattern in the air is converted into a space pattern on the basilar membrane. The nerve endings are excited in such a way that this space pattern is transferred to the brain and produces two similar space patterns in the brain, one on the left and the other on the right side. Enough of the time pattern in the air is sent to each of these stimulated patches to make times of maximum stimulation in each patch detectable, so when listening to a sound with both ears, there are four space patterns in the brain produced, each carrying also some sort of time pattern. It is a recognition of the changes in these patterns that accounts for all phenomena of audition."

THE PRESSURE SENSE THEORY OF WITTMAACK

In 1931 and 1935 Wittmaack[102, 103] presented his interpretation of the nature of the function of the end organs in the cochlea. In previous articles he had presented the hypothesis that the cristae ampullaris and maculae sacculi and utriculi do not receive their impulses by a movement of the hair of the hair cells but rather by delicate hydrostatic pressure changes in the medium surrounding the hair cells. In the articles mentioned he applies this principle to the organ of Corti. He describes the tectorial membrane at length and gives experimental evidence that this structure is extremely sensitive to hydrostatic changes in the surrounding fluid. He presents evidence that the tectorial membrane is attached not only to the hair of the hair cells but also to Hensen's cells and to the inner supporting cells, so that a series of closed cavities is formed in the organ of Corti, such as Nuel's space, Corti's tunnel and spaces between the hair of the vari-

ous hair cells as well as the internal sulcus. These closed cavities are thus separated from the rest of the endolymph. He states that the nerve fibers end nakedly or in a plexus within these spaces around the various cells. Any slight change in hydrostatic pressure in the endolymph is picked up by the delicate and hydrostatic pressure-sensitive tectorial membrane, which in turn transmits such changes to the closed spaces where the nerve terminals are stimulated.

Wittmaack refers to this as a turgor sense or hydrostatic pressure sense. There is a marked similarity between this pressure or touch concept and similar touch or pressure concepts such as presented by Hensen,[51] Waller[94] and Gray,[33] although the mechanism of transference of the stimulation is quite different.

NEWER METHODS FOR COCHLEAR STUDY

Correlation of acuity of hearing with histopathology. During the decade just passed several new lines of investigation have contributed to our knowledge of the function of the ear. One of these lines of approach was to correlate differences in the acuity of hearing as recorded by means of the audiometer with the histological findings in the ears. The most extensive studies of this type were made by Guild[36] in 1932 and again by Crowe, Guild, and Polvogt,[17] in 1934. In the latter account the authors indicate that in ears with marked high tone loss there is also marked degeneration of nerves and hair cells in the basilar turn. In less severe impairment the impairment in the cochlea is not always clearly marked. They feel, however, that this study indicates some localization of certain high tones in the basilar turn of the cochlea. They write: "Our observations definitely prove that there is some form of localization of cochlear response to certain high tones, but do not favor any particular theory, such as that of Helmholtz or of Ewald."

In 1935 Guild[37] presented further evidence relative to low tone perception by similar methods of study. After discussing the findings in five ears he writes:

> "While not clear cut, the evidence from this group of five ears indicates that even a considerable amount of nerve atrophy, when limited to the upper middle and the apical turns, does not have much effect upon the acuity of hearing for low tones; certainly the evidence does not favor the theory that there are sharply localized areas in the human cochlea for the several low tones."

And again he writes:

> "Our collection contains many examples of lesions of the lower basal turn in ears with impaired hearing for high tones, but very few examples of lesions in the upper turns in ears with poor hearing for low tones."

In 1934 Ciocco[12] in a statistical study noted some degree of localization for high tones.

Guild[35] in 1919 reported that in guinea pig ears exposed to revolver shots at close range there was, on histological examination, a marked damage to the hair cells and to the organ of Corti, usually in the middle turn with the damage fading off in the apical and basal directions.

Using the method of correlating audiograms with the histopathology of such ears, Bunch and Wolff[11] in 1935 present three patients which shortly before death had varying degrees of auditory sensitivity. The histological sections of the ears in these cases show degeneration of the organ of Corti throughout its entire extent. They state: "These findings are in agreement with those of certain earlier investigators who found that hearing was possible in the absence of the organ of Corti."

In support he cites cases from Denker and Kohler, Panse and Gradenigo. If this be so, what was nature's purpose in developing such a complicated structure as the ear.

The Wever and Bray method. The other recent method of study is based on the findings of Wever and Bray[97] in 1930. By means of an amplifying apparatus the sounds made into an animal's ear can be faithfully reproduced by placing one of the electrodes of the amplifier into the tissue of the animal and the other on the auditory nerve or on various parts of the cochlea. The impulse thus obtained has variously been described as "microphonic actions of the cochlea", "cochlear responses" or "cochlear potentials". This phenomenon has to be distinguished from "nerve action potentials" which Wever and Bray at first thought they represented.

One of the problems which investigators attempted to solve was to locate the origin of these cochlear responses. Three sources of origin have been suggested.

1.) According to the Nerve Theory of the Origin of Cochlear Responses, first suggested by Adrian, Bronk and Phillips[2] and at first adhered to by several others, it was assumed that the cochlear potentials were generated by the nervous elements within the cochlea. This theory was soon dropped, following the observation of Guttman and Barrera[38] and most of the other workers, that the cochlear response remained when the cochlear nerve was cut.

2.) According to the Hair Cell Theory of the Origin of Cochlear Responses, the cochlear potentials arise in the hair cells of the

organ of Corti. This theory rests largely on the study of congenitally deaf albino animals by Howe and Guild,[55, 56] Davis and his collaborators,[22, 71, 72] Hughson, Thompson and Witting,[58] and on damage in the cochlea by continued loud sounds as observed by Davis and his associates.[23] The objection to such conclusions is that in these cases, while there is hair cell damage, there is considerable other damage and usually there is displacement of the tectorial membrane, collapse of the cochlear duct and displacement of Reissner's membrane. Nevertheless, Stevens and Davis[93] have placed themselves in strong support of the hair-cell theory. Wever[97] has also expressed himself in favor of this theory.

3.) According to the Membrane Theory of the Origin of Cochlear responses, cochlear potentials are generated by fluid movement on the surface of, or through, semipermeable membranes within the cochlea. This theory is favored by Adrian and co-workers,[1, 2] Bast and Eyster,[6, 30] Leiri,[69] Hallpike and Rawdon-Smith[39, 40] and Ashcroft, Hallpike and Rawdon-Smith.[3]

From an anatomical standpoint one of the most serious objections against the hair cell theory and in favor of the membrane theory is the evidence of Eyster, Bast and Krasno[30] that out of 59 guinea pigs studied, the cochlear response of 23, or 39 per cent, showed no relation to the integrity of the organ of Corti. Most outstanding was the observation that good cochlear response was obtained in 5 animals with almost complete fibrosis and atrophy of the organ of Corti and the basilar membrane, and in 7 animals there was much reduced response and normal appearing organs of Corti. The other cases showed intermediate conditions.

It appears that all investigators agree that this phenomenon is not an indication of what the animal hears, but that it represents one of the events in the transmission of sound to the cochlear nerve. If all auditory mechanisms and nerve pathways and centers function normally, it may indicate what the animal hears.

COCHLEAR LOCALIZATION AS INDICATED BY COCHLEAR POTENTIALS

The evidence derived from the study of cochlear potentials by the different investigators as to localization within the cochlea is not in entire agreement, but some degree of localization is admitted by all. In general two different views on localization have been presented.

Partial localization. According to this view high frequencies are pretty definitely localized in the basal turn of the cochlea, but intermediate frequencies are much less definitely localized and there is little evidence of localization of low tones. The evidence in support of this view is presented by Bast and Eyster,[6] who found that destruction of the apical part of the cochlea caused only slight loss of low tones. Destruction of over one-half of the apical part of the cochlea did not abolish but did reduce low and intermediate tones. High tones were not affected. Hughson, Crowe and Howe[57] agree that low tones are not specifically localized and Hughson, Thompson and Witting[59] conclude:

"No evidence obtained from the procedures described above indicates localization of low frequencies to the apex of the cochlea. Though the apex is doubtless necessary for optimum experimental transmission or clinical perception of tones, any intact portion of the end organ may serve as the receptor of a stimulus of low frequency applied to the ear."

Dworkin[26] reporting incomplete results concludes: "So far as they go, these results argue against low-tone localization in the apex of the cochlea."

Wever, Bray and Horton[98] subjected animals to prolonged, intense stimulation by a specific tone. The impairment as indicated by the cochlear response was not specifically localized.

Definite localization. According to this view each pitch is localized on a specific area of the basilar membrane.

Observations in support of this view were made by Davis, Lurie and Stevens,[22, 92] who drilled holes in various parts of the cochlea destroying the specific portions of the organ of Corti. They write:

"In twenty animals a consistent correlation of this sort was obtained. High tones are localized near the basal end of the cochlea; 2000 c.p.s. is at the middle; and low tones to which we directed particular attention are bunched quite closely toward the helicotrema—more closely than has previously been supposed."

Culler and his associates[19] and Culler[18] report observations on experiments similar to those of Davis and his co-workers. Culler[18] reports two conclusions:

"(a) Each frequency within the audible range has its own focus of response within the cochlea, this focus being revealed by electric potentials which can be shown to be maximal for a single frequency at a given site.

"(b) The location of this focal point can be fixed within a maximal error of 10 or 12 percent."

THE PLACE THEORY

This theory is very similar to the Helmholtz resonance theory but instead of requiring certain specific resonating fibers, the place theory only requires that a specific tone activate a peculiar region of the basilar membrane and that there must be a certain specificity of nerve fiber action. Thus there are specific areas stimulated by high tones in the basal turn of the cochlea, while low tones have their specific areas in the apical region. Among the recent investigators, Davis and his co-workers[93] and Culler[18] are the chief adherents of this theory.

THE RESONANCE-VOLLEY THEORY

This theory was developed by Wever and Bray in recent years. The theory may be best expressed in the words of Wever:[99]

"In this theory, pitch has a two-fold representation. Low tones are represented by frequency, high tones by place, and other tones by a combination of the two. These suppositions follow the available evidence. Place alone serves for the high tones because localization in the cochlea for these is relatively specific, but the asynchronous character of nerve volleys makes frequency representation indefinite. Frequency alone serves for the low tones because they spread widely in the cochlea, yet in the nerve response they are represented with high accuracy. For the great body of intermediate tones both forms of representation are available.

"Loudness is correlated with the total number of impulses in the nerve per unit of time, and thus relates both to the number of active fibers and the rates at which they enter successive volleys. These assumptions agree with evidence on intensity representation in cutaneous and other nerves, and embrace Adrian's intensity-frequency principle."

How do we hear? This question asked by scientists and philosophers of the past must still be asked today. The conflicting observations and interpretations indicate that the problem still remains unsolved. The subject matter on this most complicated organ, the ear, has not yet been exhausted.

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XXIV

HEREDITARY HEMORRHAGIC TELANGIECTASIA

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Hereditary hemorrhagic telangiectasia is an inherited abnormality characterized by the presence of localized angiomas or telangiectases which have a tendency to bleed. The condition is well known to the internist but few rhinologists and laryngologists seem to be acquainted with it. It should be of particular interest to the latter specialists since the vascular lesions characteristic of it occur most frequently in the nasal and oral mucous membranes. Here they often give rise to severe and at times fatal hemorrhage. Numerous reports on the disease have appeared in the general medical literature but little attention has been given it in journals of otolaryngology. Quite generally the disease is considered rare and a pessimistic view is held concerning the possibility of benefit from therapy in the severe cases. As a matter of fact the condition occurs more frequently and it is of greater clinical importance than the limited number of writings on the subject would tend to indicate. Moreover in some cases it is possible with proper therapeutic measures to restore to a normal active life patients who have been invalided by the condition.

The entity was first recognized by Rendu[1] in 1896 and was elaborated on five years later by Osler.[2] Since then it has been given the names of not only these men but also the names of several other men who have written on the subject more recently. The term hereditary hemorrhagic telangiectasia, now generally applied, well describes the condition and for the sake of uniformity and simplicity its use seems desirable.

Houser[3] in 1934 presented before this Society an excellent paper on this interesting condition and directed attention to its clinical and

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Read before the Sixty-fourth Annual Meeting of the American Laryngological Association, Atlantic City, N. J., May 25, 1942.

diagnostic features, possible seriousness and methods of treatment. He reported the cases of four patients who had come under his care, all of whom were blood relations. There have been no outstanding advances in the treatment of the disease since that time, but its comparative rarity and the good results obtained with treatment in a group of cases seem ample justification for recording our experiences in this connection.

The incidence of the condition is difficult to determine since, as several other writers have previously observed, a number of the reported cases probably do not belong in this category. According to Goldstein,[4] approximately 90 to 95 families and about 500 to 550 affected persons were recorded in the literature up to 1931. It seems certain that in a good many cases the condition has gone unrecognized and that many of those in which the condition has been correctly diagnosed have not been reported so that the actual number of instances of the disease probably is considerably greater than these figures indicate. The disease affects both males and females and may be transmitted by either sex. The incidence of the condition in affected families varies greatly. Foggie[5] reported 12 persons with the condition in five generations of one family, while Hurst and Plummer[6] recorded 16 affected persons among 37 members in four generations of one family. Twenty-one members of the families cited by Paul[7] and by Steiner[8] had the disease.

During the past twenty years, 20 cases of this disease have been recognized at the Mayo Clinic. None of the patients were blood relations. Twelve of them were males, eight were females. They ranged in age from 25 to 67 years and three-fourths of them were between 30 and 60 years of age.

While the condition at times gives rise to fatal epistaxis, in general it does not inhibit development or shorten the span of life. There are no demonstrable changes in the coagulation factors of the blood and undue bleeding is not encountered after surgical procedures. In fact, submucous resection carried out in a number of these cases in an attempt to control the epistaxis has not been accompanied by unusually severe bleeding. Because of the frequent active hemorrhages, a marked degree of secondary anemia is commonly present and it is a prominent feature in all of the more advanced cases (Fig. 1). In the earlier stages of the disease, recovery from even the severe hemorrhages is rapid. Fitz-Hugh[9,10] has observed a tendency for splenomegaly and hepatic enlargement to occur in the late stages of this disease and stated that the patients exhibit an increasing intolerance to blood transfusions.

Fig. 1.—Telangiectases of the face associated with similar lesions of the nasal mucosa, palate, tongue, fingers and thorax. The patient had marked secondary anemia due to frequent attacks of severe epistaxis.

The vascular lesions characteristic of the condition are similar in appearance to the telangiectases commonly seen on the skin of normal persons, especially in later age (Figs. 2 and 3). However, histologic studies carried out by Hanes[11] and by Steiner showed extreme thinness of the walls of the blood vessels making up these angiomas. In fact, many of them were found to consist of only a single layer of endothelial cells lying immediately below the markedly thinned-out epidermis. These telangiectases occur most often about the face and especially in the nasal and oral mucosa (Fig. 5). The scalp, the ears and the conjunctiva may be involved. Next to the face in order of frequency come the neck and thorax. Other sites at times affected are the fingers, particularly the tips and the portions under the nails, also the pharynx, the larynx and the surface of various other organs of the body. The lesions commonly occur in the nasal mucous membranes during youth, but in other locations they usually do not appear until adult life is attained. The number and location of the telangiectases vary from time to time. Anemia induced by the bleeding appears to be a definite factor in this regard.

Telangiectases were present in the nasal mucosa of all the patients observed at the Clinic. The next most common sites of these vascular lesions were the tongue and the face, usually the lips and the cheeks (Fig. 4). Frequently, they were present on the dorsum

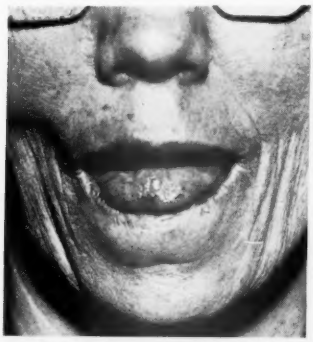

Fig. 2.—Vascular lesions of tongue and lips associated with extensive telangiectases of the nasal mucous membrane. The patient experienced numerous severe hemorrhages from the lesions in the nose but only slight bleeding from the tongue and lips.

or on the tip of the tongue. Other frequent sites of involvement were the tips of the fingers, the eyelids, ears, scalp, pharynx, palate, neck and thorax. In most instances these nevi were few in number and scattered and inconspicuous. In a few cases they were innumerable and very noticeable. They ranged from bright red to a bluish-red in color. Some of them were flat but in many of them the center was slightly elevated above the surface of the surrounding skin. As Osler[12] had observed, bleeding seemed more likely to occur from those whose midportion was raised since these were most subject to trauma.

In the nose the telangiectases were situated most often on both sides of the cartilaginous portion of the septum. This probably accounted for the fact that, in many of the patients in whom the condition had existed for a number of years and in whom repeated cauterizations of the lesion had been carried out, a large septal perforation was present in this region. Vascular networks were also commonly present in the mucous membrane of the lateral nasal walls, particularly on the inferior turbinates. All of these angiomas situated intranasally were more readily traumatized and they bled more freely than the lesions on the cutaneous surfaces or those inside the mouth.

Epistaxis is the most common symptom of the disease, and it is usually because of this that the patient seeks medical advice. The bleeding varies greatly in frequency and severity. It may be an oozing, recurring at frequent intervals or persisting continuously for several days. On the other hand, there may be profuse hemorrhages

initiated by sneezing, coughing or any slight strain or coming on spontaneously while the patient is lying quietly in bed. These hemorrhages may occur only at intervals of two or three weeks or they may be repeated four or five times daily. Several hundred cubic centimeters or more of blood are frequently lost in a single hemorrhage and the patient is often markedly weakened as a result.

The tendency to bleed is greatly increased in these individuals by active movement and often it is impossible for them to do any manual labor or even to mingle socially. Next to the telangiectases in the nasal mucous membrane, bleeding most frequently occurs from those in the oral mucosa. It may take place from such lesions situated in almost any epithelial or mucous surface of the body. It has been observed from various portions of the cutaneous surface including the face, ears, scalp and finger tips, also from the lips, tongue, pharynx and larynx. It has been reported from different parts of the gastro-intestinal tract, particularly the stomach and rectum, from the genito-urinary system, the trachea, bronchi and meninges, but the points of bleeding in these situations were not definitely proved to be telangiectases. Essential hematuria has been thought to be on this basis in a number of instances. The bleeding tendency is prone to increase toward middle life and it is aggravated by severe anemia.

In all of the cases of this condition coming under our observation epistaxis was the presenting complaint. Most of the patients had been troubled with this since childhood and a number stated that they had had it all of their lives. In only two cases was it of short duration, only one year. In one of these cases the patient was a woman 35 years of age; in the other the patient was a woman of 65 years. The latter patient gave a positive family history, and telangiectases which had bled at times were present on her fingers as well as in the nasal mucosa. Eight patients had experienced hemorrhages from telangiectases in areas other than the nasal fossae, among them the lips, tongue, face and fingers. One was having bright blood in the stools associated with diarrhea. Proctoscopic investigation of this was not carried out. The severity of the nasal bleeding varied greatly, ranging from a persistent oozing, which at times continued for as long as four days, to profuse hemorrhages lasting for one-half hour or more in spite of packing or other measures. In most of the cases the severity of the bleeding had increased in recent years. Some of the patients had been so weakened by repeated hemorrhages that they had been unable to carry on any manual labor for periods of several months to two or three years. In a number of instances, repeated transfusions had been required. The frequency of the bleeding varied in different cases and

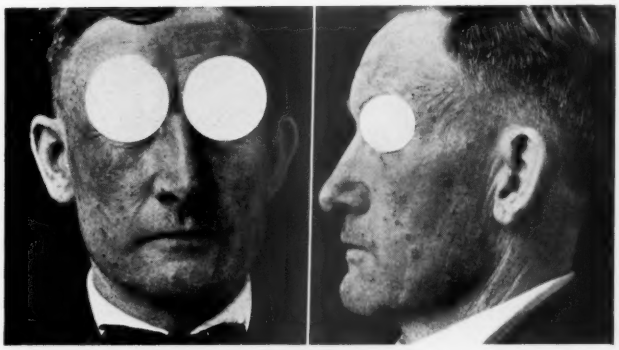

Fig. 3.—Hereditary hemorrhagic telangiectasia with unusually large vascular lesions of the face of a man 49 years of age. Similar angiomas were present on the scalp, tongue and inside the nose. The patient wrote nine years later that the lesions had greatly increased in number.

in the same case at different times. Some of the patients experienced epistaxis several times monthly or weekly, while in others active nasal bleeding occurred from one to six times daily.

Larrabee and Littman[13] said that a diagnosis of hereditary hemorrhagic telangiectasia can be made only when a definite history of heredity is obtainable and when multiple telangiectases that have a tendency to bleed are visible about the face and nose. However, Fitz-Hugh has clearly shown that atavism unquestionably exists in this connection so that, although the disease is hereditary, it may not be manifest in the immediately preceding generations. Accordingly, a history of heredity may be difficult, if not impossible, to elicit, yet the clinical features may be so definite that a diagnosis is warranted. From personal observations we are convinced that the clinical history and findings are often sufficiently clearcut to permit of positive recognition in the absence of known familial involvement. Six of our patients knew of no members of their families who had had similar trouble, but some of the patients had been out of touch with their families for years and had very little knowledge concerning them.

DIAGNOSIS

From the standpoint of diagnosis, hemophilia, purpura hæmorrhagica, aplastic anemia, and acute leukemia must be considered. Hemophilia occurs almost exclusively among males and is trans-

mitted by females. Hemarthrosis, accompanied by pain, is a common symptom. From the laboratory standpoint, the bleeding time and platelet count are usually within normal limits and the clot retraction time is usually normal but may be slightly prolonged. The characteristic laboratory finding is usually a marked prolongation of the coagulation time of the blood. In purpura hæmorrhagica the patient usually presents a history of cyclic episodes of hemorrhage associated with numerous petechiae and large ecchymoses over the body and particularly over the extremities. These hemorrhagic episodes may be of very minor character and of short duration although in certain cases a history of earlier episodes of hemorrhage cannot be obtained. The blood platelets are reduced in number, the bleeding time is prolonged and there is markedly delayed or absent clot retraction whereas the coagulation time is usually within the normal limits or only slightly prolonged. In aplastic anemia there is usually a history of weakness and anemia prior to the onset of hemorrhage. The spleen is not palpable and the coagulation time usually is normal whereas the platelet count is reduced, the bleeding time is prolonged and the clot retraction delayed. Acute leukemia may be suspected from a history of rapid onset of anemia, fever, ulceration of the mucous membranes and hemorrhagic manifestations as the disease progresses. The spleen is usually palpable since there is frequently a reduction in platelet count with consequent lengthening of the bleeding time and delay of clot retraction. The leukocyte count in acute leukemia is usually well above normal but in many instances the count may be normal or a marked leukopenia may be present. Examination of the blood smears is of value in the differential diagnosis. In hemophilia, if there is active hemorrhage there will be increased erythrocyte regeneration with the general appearance of the smear otherwise essentially normal, although a slight leukocytosis may be present. In purpura hæmorrhagica, a marked reduction in the platelet count associated with evidence of active bone marrow regeneration will be seen, whereas in aplastic anemia evidences of a decrease in bone marrow activity are readily seen. In acute leukemia, the presence of very immature leukocytes in the peripheral blood establishes the diagnosis. In hereditary hemorrhagic telangiectasia, the blood coagulation factors are within normal limits, the telangiectases are most frequently seen on the face, neck and trunk, are bright in color, often elevated and tend to partially fade on pressure.

TREATMENT

The different therapeutic measures advocated for dealing with this condition aim at producing symptomatic relief only. The disease

Fig. 4.—Telangiectases of the external portion of the nose and of both surfaces of the lower lip associated with similar lesions in the nasal mucosa. The lesions in the nasal mucosa were producing severe epistaxis. The patient was a woman, 38 years of age.

Fig. 5.—Lesions of hereditary hemorrhagic telangiectasia involving the tongue and palate of a man 62 years of age.

is due primarily to a defect of the walls of the capillaries and venules, and therapy is not likely to benefit this.

Among the therapeutic measures previously employed in the case of patients who later came under our care were radium, actual cautery, local applications of caustics, particularly silver nitrate, vitamin K, application of roentgen therapy to the spleen, moccasin snake venom, intranasal surgical procedures, especially submucous resection, transfusions, and the internal administration of various preparations of iron. One patient had received 15 or 16 blood transfusions. Another had been packing his own nose with gauze when the bleeding was severe. In most of the cases the bleeding tendency, especially the epistaxis, was becoming worse in spite of treatment.

For the immediate control of hemorrhage by the patient the most useful device is the ingenious modification of the Cooper Rose inflating plug devised by Hurst and Plummer. This consists of a finger cot placed over the end of a small catheter and tied snugly with fine thread. This is kept continuously at hand by the patient so that when active bleeding occurs it can be lubricated, inserted well back in the nostril and inflated either by placing the open end of the catheter in the mouth or by means of a rubber bulb. Firm uniform pressure is thus applied to the entire interior of the nasal fossa and will usually effectively control the bleeding for the time being. After the hemorrhage has stopped the cot is slowly deflated and withdrawn or allowed to drop out of the nostril.

For more lasting effect, radium, actual cautery, electrocoagulation and various chemical caustics, including chromic acid, trichloracetic acid and others, have been employed. Houser stated that chromic acid proved more effective than other types of cautery in his hands. In our practice, electrocoagulation has given better results than any other form of therapy. This has usually been carried out following cocainization of the nasal fossae but in a few instances general anesthesia has been necessary because of the patient's inability to tolerate the discomfort associated with it. For this purpose, pentothal sodium has been administered intravenously. The coagulation requires extreme patience, persistence and gentleness. Before using it, the bleeding should be controlled with liberal applications of epinephrine and a 10 per cent solution of cocaine, and the site of the points of hemorrhage must be carefully noted. In the face of active bleeding, the current is likely to be dissipated so rapidly that coagulation progresses very slowly and when it does take place excessive adherent crusting forms. As the electrode is then moved, the bleeding point

is reopened. Often this will occur repeatedly and only persistent, thorough electrocoagulation will control it. Frequently, too, after one has checked the bleeding at one point it will become active at another point well removed until one is tempted to insert a pack rather than spend more time. However, this usually is not a wise policy for even vaseline gauze is likely to adhere sufficiently so that further hemorrhage will develop upon its withdrawal.

In view of the intensive electrocoagulation required in some of these cases, it is difficult to conceive of chromic acid or other similar chemical caustics effectively controlling the bleeding. At times, as the current is applied the spark from the electrode will immediately perforate the thin wall of the vessel and produce active hemorrhage that is difficult to control. In a few instances, in which hypertension was associated with the primary condition, the stream of blood emitting from this perforation extended entirely across both nasal fossae from one lateral wall through the large perforation in the septum against the lateral wall of the opposite side. It was found by experience that a relatively high voltage with low amperage was most satisfactory in coagulating the telangiectatic lesions. When the spark gap of the diathermy machine was widened, there was a much greater tendency for the spark to perforate the thin-walled vessels and induce severe bleeding. On a number of occasions it has been necessary to use diathermy persistently for one and a half or two hours at a single sitting except for periods of intermittent cocainization, but the ultimate results have well repaid the effort.

Houser noted that in one of his cases, regardless of how thoroughly the nevi were destroyed, new ones promptly formed in the adjacent mucosa and these showed just as much tendency to bleed as did the original lesions. This was observed in several of our cases and was a most discouraging feature, for at times the patients' economic situations did not permit their remaining under prolonged observation or returning later for treatment. At times, too, because of this the patients became convinced that the situation was hopeless and stopped the treatment. However, in the case in which this tendency was most pronounced and in which severe disability had occurred, our most brilliant result was obtained. The patient, a woman, 52 years of age, had suffered from severe nasal hemorrhages for fifteen years, for which radium and various other forms of treatment had been applied locally. As a result, most of the nasal septum had been destroyed but the epistaxis had continued. The bleeding had, in fact, been so severe that the patient had been invalided by it and had been bedridden for several weeks at a time. Electrocoagulation was carried

out repeatedly during the course of several years, and as a result of the scarring-down of the telangiectases produced by this the patient now rarely experiences any epistaxis and is able to enjoy a perfectly normal life. In a recent letter, approximately eight years after her first coming under our care, she stated that the last four years have been the best of her entire life.

About the lips, the oral cavity, and on the cutaneous surface of the body, the electrocoagulation has proved even more satisfactory in eradicating the angiomas than in the nasal fossae. In some of the cases in which intensive intranasal coagulation has been necessary, it has been feared that collapse of the nasal bridge would result and the patients have been advised of this possibility. To date, this has not occurred, however, and as the process has now been quiescent for upward of from one to four years in these cases, it is not anticipated that deformity will take place.

The use of radium in treating this condition is in our opinion contraindicated. While it may result in definite improvement for a time, it invariably produces marked atrophy and dryness of the nasal mucosa later and the associated crusting greatly increases the tendency to bleed. There is a possibility, too, that the telangiectases which are prone to develop following radium therapy may increase the hemorrhagic tendency. As a matter of fact, dislodgment of crusts has seemed to be a most important factor in inducing hemorrhage in these cases generally, and liberal local applications of mineral oil or petrolatum together with iodides internally at times are beneficial. In one of our cases, in which the patient had been progressing satisfactorily under electrocoagulation but who, for economic reasons, was unable to remain for continuation of this, further bleeding developed following his return home. He had previously received radium treatment and in spite of his own protests further irradiation was insisted upon. The bleeding became more severe and, according to a letter received from his daughter, he died of hemorrhage while the treatment was being given. It seems probable that the bleeding in this case might well have been controlled with diathermy and a fatal termination averted for some time at least.

Transfusions of citrated blood are often essential in combating the secondary anemia associated with this condition, but they exert no curative effect. We have not observed the increasing intolerance to transfusions in these cases described by Fitz-Hugh. The use of snake venom as a therapeutic measure in this connection has been disappointing. One patient reported that he was unable to tolerate it and another stated that his bleeding tendency was worse since using it. Only one patient to whom it was administered felt that it had been

beneficial. This individual reported, a year subsequent to treatment at the Clinic, that his telangiectasia was not progressing.

SUMMARY

This report is based on 20 cases of hereditary hemorrhagic telangiectasia seen at the Mayo Clinic. Epistaxis was the presenting complaint in all of the cases. Electrocoagulation was found to be the most effective means of controlling the nasal hemorrhages. Snake venom proved to be of little benefit in these cases.

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XXV

THE ANTERIOR COMMISSURE TENDON

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It is common knowledge gained from operative experience that one of the earliest and most frequent break-throughs of carcinoma of the larynx is in the anterior midline. There are two factors that are probably the cause of this phenomenon: (1) Carcinoma occurs in the anterior portion of the larynx in a great majority of cases; and (2) carcinoma of the larynx is of a squamous cell type (95 per cent), one that destroys by direct extension rather than by metastasis.

Any weak spot or deficiency in the cartilaginous cage may be penetrated by a new growth occurring in close proximity. A subglottic growth may penetrate the cricothyroid membrane, another weakness is to be found in the posterior wall of the crista of the thyroid cartilage where the anterior commissure tendon is inserted.

This tendon is a band of fibrous tissue containing lymphatics and blood vessels and dividing in the center to attach to each side of the muscular larnyx. The attachment of the tendon extends from the upper portion of the thyroid cartilage caudalward for approximately 10 mm. and is about 1 mm. in width. Serial cross-sections of the anterior commissure region of a normal larynx demonstrate the firm attachment of the anterior portion of the muscular larynx into the midline of the posterior surface of the thyroid cartilage. The blending of the fibrous tissue of the anterior commissure tendon into cartilage is shown in the above-mentioned illustrations. In the lower portion of the larnyx, the tendon sweeps around with no firm attachment to the cartilage. Though the tendon is firmly attached to the thyroid cartilage for most of its extent, the internal perichondrium on each side of the tendon attachment is not firmly attached to the cartilage and is easily separated.

Since the anterior attachment of the muscular larnyx is firm and the lateral attachment is loose, a dorsal pull would seem to be the

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Read before the Sixty-fourth Annual Meeting of the American Laryngological Association, Atlantic City, N. J., May 25, 1942.

Fig. 1

Fig. 2

Fig. 1.—Cross section of the anterior commissure region of a young adult larynx above the false cords (X 20). *1*, Attachment of anterior commissure tendon; *2*, thyroid cartilage; *3*, perichondrium; *4*, ciliated epithelium; *5*, numerous glands.

Fig. 2.—Section approximately 3 mm. lower (X 20). *1*, Anterior commissure tendon; *2*, anterior commissure (squamous epithelium) (ciliated epithelium in folds); *3*, perichondrium.

Fig. 3

Fig. 4

Fig. 3.—Section approximately 6 mm. lower (X 20). *1*, Anterior commissure tendon; *2*, anterior commissure (squamous epithelium); *3*, mass of elastic tissue; *4*, perichondrium.

Fig. 4.—Section approximately 9 mm. lower (X 20). *1*, Anterior commissure tendon; *2*, anterior commissure (squamous epithelium); *3*, muscle fibers; *4*, mass of elastic tissue; *5*, perichondrium.

Fig. 5

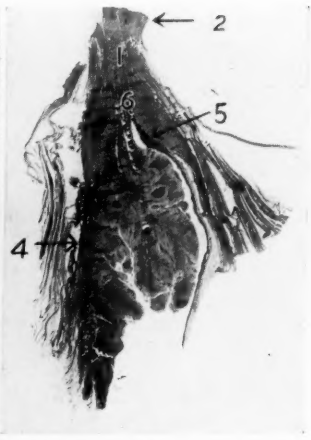

Fig. 6

Fig. 5.—Section approximately 12 mm. lower (X 20). *1*, Anterior commissure tendon, no fixation into thyroid cartilage; *2*, perichondrium almost continuous; *3*, mass of elastic tissue; *4*, anterior commissure (squamous epithelium).

Fig. 6.—Cross section of the anterior commissure region of a male, 62 years old; early carcinoma, left vocal cord (X 10). *1*, anterior commissure tendon; *2*, thin strip of thyroid cartilage; *3*, mass of elastic tissue; *4*, carcinoma extending deep to perichondrium; *5*, hyperplasia of mm. on opposite cord; *6*, anterior commissure (in anterior ciliated epithelium, in posterior squamous epithelium).

main force exerted in this region. Also of interest is the mass of elastic tissue just posterior to the division of the tendon and located in the anterior tip of both vocal cords.

The only notation of this tendon found was made by Ridpath,[1] in a description of the thyroid cartilage. He states, "situated in the median line and on the inner surface we find a small fibrous projection which serves for the insertion of the vocal cords."

In all probability, early recurrences in cases of carcinoma of the anterior larynx which were removed through a midline incision of the thyroid cartilage should be considered continuations rather than recurrences, and as a carcinomatous growth which was retained in this tendon or in its insertion into the thyroid cartilage.

In those cases of carcinoma of the larynx where the growth has invaded the tendon and perhaps the cartilage, it would seem advisable

to either perform a laryngectomy or remove the midportion of the thyroid cartilage along with the diseased cord.

Appreciation is due to Miss Mary Ramsay of the Department of Pathology for the preparation of the sections.

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XXVI

THE KELLY OPERATION FOR RESTORATION OF LARYNGEAL FUNCTION FOLLOWING BILATERAL PARALYSIS OF THE VOCAL CORDS. REPORT OF THREE CASES.

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Increasing interest has been manifested in the restoration of laryngeal function following bilateral paralysis of the vocal cords within the past three years, because of new operative procedures presented by King[1, 2] in 1939 and Kelly[3] in 1940. The operations devised by these men have resulted in the greatest number of successfully operated cases yet obtained in efforts to restore the function of the larynx after the development of this distressing condition.

Injury to the recurrent laryngeal nerves during thyroid operations is considered the primary cause of bilateral paralysis. Injury to one nerve may produce this condition when there is a pre-existing paralysis of the opposite cord. A small percentage of cases are caused by central lesions. Fortunately, their occurrence is relatively uncommon.

It is generally accepted that the recurrent laryngeal nerve supplies all the intrinsic muscles of the larynx with the exception of the cricothyroideus, which is supplied by the external division of the superior laryngeal nerve. This muscle has long been considered as a tensor of the cords but is now thought by Lemere[4], Clerf[5] and other prominent laryngologists to have the additional function of adduction, which is thought responsible for the eventual midline position of the vocal cords following injury to the recurrent laryngeal nerves.

There are three types of cases which may be encountered:

1. A small percentage of cases in which, immediately following injury or sectioning of the recurrent nerves, the vocal cords assume a midline position and are so closely approximated that marked difficulty and stridor develop.

Read before the Southern Section meeting of the American Laryngological, Rhinological and Otological Society, Chattanooga, Tenn., Jan. 28, 1943.

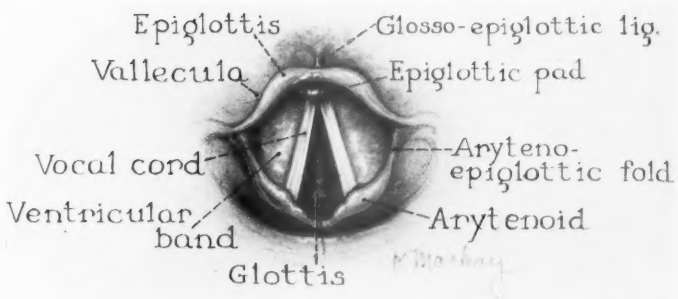

Fig. 1.—Normal larynx on inspiration.

2. Cases in which the vocal cords assume an intermediate position, midway between abduction and adduction, often referred to as cadaveric, and never adduct to such an extent as to interfere with breathing, though the voice is forever impaired.

3. Cases, which constitute a majority, in which the vocal cords first assume an intermediate position, signified by marked voice changes but no interference in breathing, and gradually return to the midline position. The degree of distress depends upon the patency of the glottis, which is usually only sufficient for the maintenance of life but not for much activity.

The first consideration in therapy is to relieve these individuals of their agonizing efforts to obtain a sufficient amount of air. This is readily accomplished by a low tracheotomy. In the first group of cases tracheotomy should be performed as a life-saving measure immediately following the development of marked stridor and impending asphyxia. The second group of cases requires no interference, while in the third group tracheotomy should be performed whenever respiratory difficulty develops on the slightest exertion or when breathing becomes stridorous at night. This may vary from a few weeks to several months.

After the individual has become acclimated to the tracheotomy tube and has recovered his sense of well-being and health, he soon tires of wearing the tube and of its accompanying inconveniences and begins to seek other methods of correcting the disability.

Kelly, in search of the solution of this problem, whereby an adequate laryngeal airway could be obtained, a normal or good speaking

Fig. 2.—Vocal cords in normal phonation.

Fig. 3.—Bilateral abductor paralysis of the vocal cords. There is only sufficient glottic space for limited activity. The voice is good.

Fig. 4.—Paramedian position of the vocal cords in bilateral abductor paralysis. There is no disturbance in breathing. The voice hoarse and poor.

Fig. 5.—Bilateral paralysis with the vocal cords in cadaveric position. There is no disturbance in breathing; a hoarse whispered voice. This is an infrequent finding.

voice maintained and the tracheotomy tube dispensed with, reviewed all previous methods and operative procedures which had been performed for this disability. These methods consisted of suturing the severed ends of the recurrent nerves[6], nerve anastomosis[7-10], transplants of the anterior extremities of the vocal cords[11], cordectomy or ventriculocordectomy[12], all of which failed to accomplish the desired results. Submucous resection of the vocal cord with the removal of the vocal process of the arytenoid by Hoover[13], in 1932, and more extensive submucous resection with complete removal of the arytenoid cartilage by Lore[14], in 1936, offered more promising results. The King operation, presented in 1939, offered the most successful results obtained up to that time. This operation was directed toward the exterior of the larynx and consisted essentially in mobilizing the arytenoid cartilage and displacing it outward by suturing it to the wing of the thyroid cartilage in such a manner as to abduct the vocal cord to the desired position. He first transposed the omohyoid muscle to the arytenoid, thinking this would give motility to the cord, but later considered this relatively unimportant.

Kelly[3] concluded that the best results were obtained where operative procedures were directed to the exterior of the larynx (thus eliminating any possibility of intralaryngeal cicatricial stenosis) and where the arytenoid was involved. He conceived the idea of completely removing the arytenoid cartilage by an external approach, through a window made in the wing of the thyroid cartilage directly over the arytenoid. His idea was that he would destroy the tension of the cord on the operated side and allow it to fall away from the midline. Also, he felt that the space created by the loss of the arytenoid would be obliterated by nature, that atrophy of the muscles would occur on the operated side and there would thus be a further increase in the area between the cords posteriorly. He felt that there would be only slight alteration in the position of the anterior half of the cord and a serviceable voice would thus be obtained.

The operation should be performed only in those cases in which bilateral abductor paralysis has existed for six months or longer. It is seldom that function of the cord returns after six months, though a few cases have been reported. A tracheotomy should be performed if this has not been performed previously.

Intratracheal ether anesthesia has been found to be the most suitable, using a Flagg intratracheal tube, size 5 to 7 mm. The intratracheal tube aids in fixing the arytenoid cartilage while it is being dissected and does not interfere with the field of operation. Anesthesia

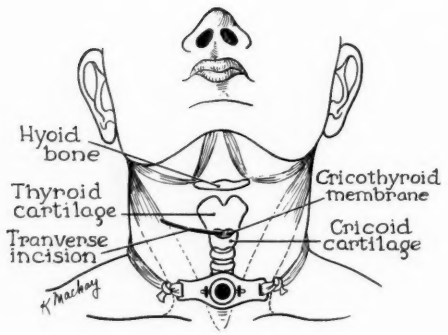

Fig. 6.—Important structures in midline of the neck. Initial transverse incision for arytenoidectomy extending along the right lower border of the thyroid cartilage to the sternocleidomastoid muscle.

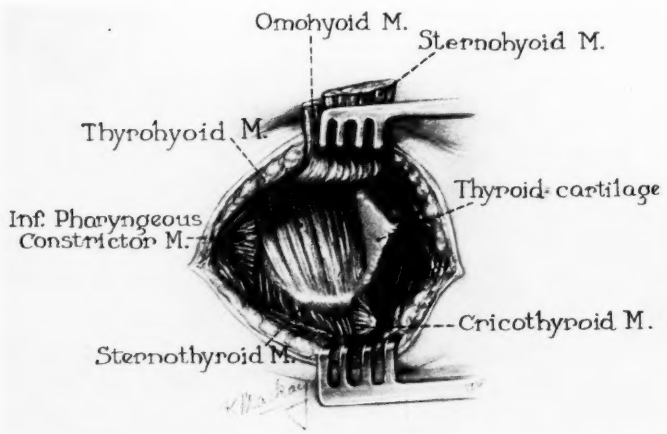

Fig. 7.—The pretracheal and laryngeal muscles exposed. The sternohyoid and omohyoid muscles have been sectioned and reflected bringing into view the sternothyroid and thyrohyoid muscles.

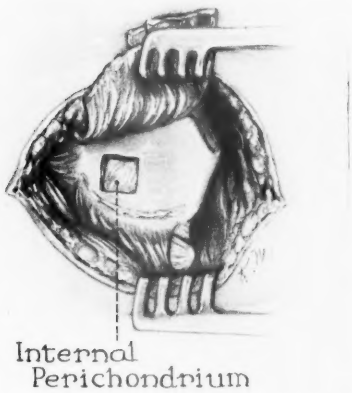

Fig. 8.—The right wing of the thyroid cartilage has been exposed by reflection of the sternothyroid and thyrohyoid muscles. A window has been cut in the lower outer two-thirds, 1 to 1.5 cms. square, through the external perichondrium and cartilage, down to the internal perichondrium. The cartilage is removed with small rongeurs.

may be started through the tracheotomy tube and the intratracheal cannula be inserted after anesthesia is complete. The tracheotomy tube is then removed.

The head and neck are then slightly hyperextended by placing a small sandbag or rolled sheet, prepared as for any external laryngeal operation, under the shoulders and the neck. The thyroid and cricoid cartilages are carefully palpated in the midline for proper orientation. A transverse incision, about 3 in. long, is then made along the lower border of the thyroid cartilage, through the superficial structures and the platysma muscle and extending from just across the midline to the medial margin of the sternocleidomastoid muscle, on the side elected for operation. The skin flaps are then retracted bringing into view the pretracheal muscles which are the sternohyoid and the omohyoid. The muscles are severed and reflected, bringing into view the underying sternothyroid and thyrohyoid muscles, which in turn are elevated from the wing of the thyroid cartilage. The arytenoid cartilage may now be palpated by rotating the larynx to the opposite side and placing the forefinger beneath the lateral border of the wing

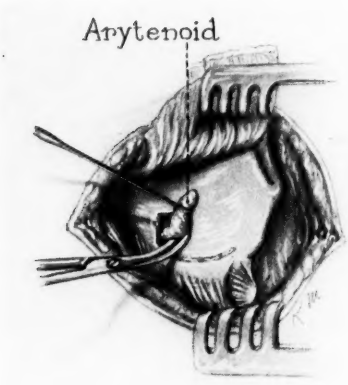

Fig. 9.—The aryteroid cartilage being delivered through the window after elevating the internal perichondrium and dissecting the arytenoid cartilage free from its muscular attachments.

Fig. 10.—Laryngoscopic view of submucous resection of arytenoid cartilage. A small hook may be seen passing through the window beneath the mucous membrane and engaging the arytenoid cartilage.

of the thyroid. No major vessels or important structures are exposed throughout this procedure.

A window, about 1 to 1½ cm. square, is then made with a scalpel in the lower lateral two-thirds of the wing of the thyroid down to the internal perichondrium, and about 2 mm. from the lateral and lower margin of the thyroid. The cartilage within the window is then removed with a small rongeur and curette, exposing the internal perichondrium. The window may be enlarged later if necessary. The internal perichondrium is incised on three sides and elevated upward, exposing the thyro-arytaenoideus muscle. Slight bleeding is usually encountered but is readily controlled with small adrenalin packs or the injection of a few minims of adrenalin into the muscle. A small hook is then introduced through the muscle fibers and moved gently about until the arytenoid cartilage is located. The cartilage is engaged and braced against the intratracheal cannula. Small, slightly curved Stevens scissors are then introduced and passed posteriorly where the cricoarytenoid articular capsule is incised. With a slight pull on the hook, the arytenoid can be felt to give as the articulation is severed. By constantly exerting slight tension on the arytenoid with the hook, the cartilage is completely freed from its muscular attachments; the scissors should be kept close to the cartilage at all times. As the cartilage is separated from its attachments, it is gently worked upward through the window until completely delivered.

This is the most delicate part of the operation and requires considerable patience. Care must be exercised not to perforate the larynx or cut the cartilage, for it is then much more difficult to remove.

When the arytenoid has been removed, the intratracheal cannula is withdrawn and the larynx exposed with a direct laryngoscope. With an associate grasping the posterior extremity of the cord through the window, the excursions made by movement of the cord can be readily visualized. In this way the degree of abduction of the cord and the resulting improved glottic space can be easily determined.

It was Kelly's impression that contraction, resulting through the normal processes of healing, would provide sufficient abduction of the vocal cord and thus create ample glottic space for normal activity. This has proven to be true but in certain cases the individual is apt to be slightly handicapped on excessive exertion, which invariably occurs at times during the course of an individual's normal routine.

To secure greater space, which would be sufficient for most all requirements, the extreme posterior end of the vocal cord may be sutured to the external perichondrium at the lower margin of the

Fig. 11.—Postoperative right arytenoidectomy. The posterior extremity of the vocal cord has been sutured in the abducted position, creating sufficient glottic space for normal breathing and maintaining a good speaking voice. Direct laryngoscopy.

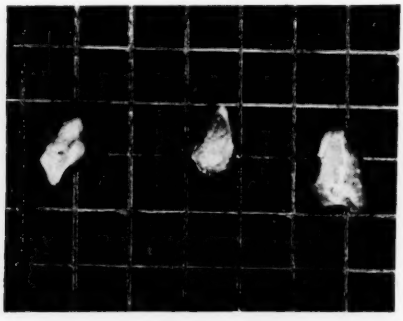

Fig. 12.—Arytenoid cartilages removed in three cases.

window with triple zero chromic cat gut in any desired position. Abduction of the cord to 6 or 7 mm. from the midline seems desirable and affords sufficient breathing space without sacrificing the voice to any great extent. It must be remembered that every millimeter increase in abduction of the cord will result in greater alteration of the voice and the ultimate objective is only to secure an adequate glottis and preserve a good voice. I believe that placing a suture and fixing the cord in abduction is of the utmost importance and should be performed in all cases.

The wound is then closed in layers and a rubber tissue drain inserted in one end. A nasal feeding tube is introduced, the tracheotomy tube reinserted and a flat gauze dressing applied.

Complications are not common. If the laryngeal mucosa has been perforated and blood escaped into the trachea, it should be immediately aspirated with a catheter. Excessive tracheal or bronchial secretions following operation should also be aspirated as indicated. If infection should develop sulfonamide therapy should be instituted. There is usually considerable edema of the arytenoid processes and aryepiglottic folds, but normally this should practically subside within five to seven days. When the edema has subsided sufficiently to allow easy swallowing, the feeding tube is removed. In certain cases there may be very little edema and the feeding tube may be removed within 48 hours. The sutures are removed on the fourth or fifth day and the patient allowed to be up.

Within a few weeks there is noticeable improvement in breathing, but the tracheotomy tube should not be removed until breathing is free and easy at all times with the tracheotomy tube occluded. This may require several weeks or several months, depending on the size of the glottis and the psychological reaction of the patient. If the vocal cord has been transfixed, decannulation can be accomplished at a much earlier date.

Kelly has now had 25 operative cases with 20 successful results. He recommends that if results are not satisfactory following arytenoidectomy on one side, the operation can be performed on the other side, or the original window may be re-exposed and the vocal cord further abducted.

I would like to report three cases which have been under my observation and operated upon by this technic. All three patients were females in their late thirties or early forties, had had previous thyroid operations and had worn tracheotomy tubes from one to eight

Fig. 13.—Colored patient who has been wearing tracheotomy tube for eight years. Tracheostomy before closing.

Fig. 14.—Same patient as shown in Fig. 13. Decannulated one month after arytenoidectomy. Tracheostomy after closing.

years. The colored patient required emergency tracheotomy on the operating table at the time of a thyroid operation. The other two cases submitted to tracheotomy only after months of difficult breathing.

Arytenoidectomy was performed on the right side in two cases and on the left side in the other. In the third case there was very slight motility of the right cord and it was deemed advisable to operate upon the immobile side. The postoperative course was normal in all cases.

The first patient operated upon in this series obtained noticeable improvement in breathing after a few weeks, but reached maximum improvement only after four months. She has adequate breathing space for normal activity but has slight difficulty in breathing on excessive exertion, and has not yet been decannulated. This is primarily because of psychological reasons as she has kept her tracheotomy tube occluded constantly, except when asleep, for the past four months. This patient was especially concerned about her voice, being a business secretary, and her vocal cord was not transfixed. Her voice is now excellent though slightly less clear than normal. I feel that her tube can be safely removed at any time.

The second patient has had an unusual complication which prevents any noticeable improvement. There has been a persistent edema

of the laryngeal vestibule which I have been unable to explain. This has gradually subsided but as yet, eight months after operation, it is impossible to visualize her vocal cords by indirect laryngoscopy. There was similar prolonged edema of the larynx following tracheotomy, which was most unusual, and it appears that there must be some disturbance of the laryngeal lymphatics, which require an excessive time for readjustment following manipulation about the larynx.

The colored patient, my first operative case and performed shortly after assisting Dr. Kelly, obtained excellent results. The vocal cord was sutured to the external perichondrium in this case, creating a permanent glottic space of about seven millimeters. Adequate breathing space was obtained and a good speaking voice maintained. She was decannulated one month following operation after having worn a tracheotomy tube for eight years.

In conclusion, I would like to call attention to the importance of laryngeal examination preceding and following thyroid operations. The diagnosis of bilateral abductor paralysis can easily be determined by indirect laryngoscopy. Previous attempts at correcting this distressing condition have met with little success, until the King and Kelly operations were devised. These offer a corrective procedure and assure an adequate glottis and speaking voice. The Kelly operation appears to be the simpler and more easily performed of the two procedures.

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XXVII

THE PROBLEM OF ACUTE CATARRHAL OTITIS MEDIA

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AND

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About two years ago my associate, Dr. Hoople, and I were discussing the problems of treatment incident to our practice, and among other things we talked over the problem of acute catarrhal otitis media. It seemed to us that present day methods of treatment for this condition are essentially the same as they were fifty or sixty years ago, and that, in general, the whole course of treatment for these patients is rather discouraging. We decided to check over our results and attempt to evolve a rationale of treatment on a sound scientific basis if possible.

In this paper there will be nothing that is startlingly new and different in the way of treatment. It is essentially a recapitulation of some of the known facts and an inventory of our knowledge and opinions. The problem of treatment of acute catarrhal otitis media is still, to our minds, one of the unsolved problems of otolaryngology.

The signs, the symptoms, and the pathology of this condition are well known to all and need not concern us at the present.

In reviewing our case histories I found that 804 patients had been treated for acute catarrhal otitis media over a period of 15 years. Of these, 453 were females and 351 were males. The average age of the patients was 28 years.

The right ear was involved in 233 patients, and of these, 210 had fluid or serum present in the middle ear. The left ear was involved in 240 patients, with fluid present in 209. Both ears were involved in 372 patients.

Presented before the Eastern Section Meeting of the American Laryngological, Rhinological and Otological Society, Hartford, Conn., Jan. 15, 1943.

The method of treatment of these cases consisted for the most part in treating the infections of the nose and throat, when such could be found, followed by politzerization or catheterization of the eustachian tube and inflation of the middle ear. Whenever fluid was present, paracentesis of the drum membrane was carried out as well. This latter procedure was done in about one-half of the cases (393). Some received bouginage of the eustachian tube, some required aspiration of the middle ear by suction, and a small number received x-ray to the nasopharynx, insulin or thyroid, and various other types of treatment.

It is somewhat difficult to evaluate accurately the results of treatment in these cases. The average number of treatments per patient was six. This figure, however, does not give a proper indication of success or failure, for many patients were treated only once or twice and then did not return. Others were treated numerous times over a period of months and still were not entirely free from symptoms. Some of the cases included in this series undoubtedly fall into the class of subacute or even chronic catarrhal otitis media, and to draw a definite line where the acute condition leaves off and the chronic begins, at least from the clinical standpoint, is impossible. In the series of 804 cases, 15 were definitely not improved after considerable treatment and numerous inflations of the middle ear.

In looking for predisposing factors in the cause of acute catarrhal otitis media, a few interesting facts appear. Six hundred and sixty-six patients of the 804 had infections of the nose or throat just prior to the onset of the ear symptoms, and in eight cases the ear difficulty arose as a result of a contagious disease.

Homolateral maxillary sinusitis was present in 44 cases. Acute tonsillitis and an infection in the adenoid tissue occurred in 50 cases. Acute ethmoiditis was present in 71 and acute sphenoid sinusitis in one case. One hundred and sixteen patients showed a marked deviation of the nasal septum to the affected side.

One hundred patients in the group showed no evidence whatever of infection, either before or during the course of the ear involvement. This group showed the following conditions:

Allergy		68
Endocrine problems:	Hypothyroid	19
	Hypopituitary	2
Pregnancy		2
Aero-otitis		7
Trauma		2

Perhaps a few words of explanation concerning this group would not be amiss. The 68 patients with allergy were almost entirely victims of hay fever and their ear symptoms occurred at the height of the ragweed season when the nose was almost completely blocked. The endocrine cases were fairly obvious. Most of these were referred back to their own physicians for complete check-ups including basal metabolic tests. As a result 19 received thyroid in appropriate dosage, even as high as six grains per day. In all these cases the ear situation improved shortly afterwards. The two cases recorded as hypopituitary problems were referred back to one of the local obstetricians, for both of these were women who had been under his care for delivery some months earlier. It was at his suggestion that A. P. L. hormone was used in both instances, and apparently with excellent results, for the ear condition rapidly improved after having been static for several weeks.

The two patients who developed acute catarrhal otitis media during pregnancy showed symptoms during the last trimester of pregnancy when the nose was particularly engorged. Both cleared up completely shortly after delivery. It is interesting to note here that one of these patients is again pregnant and again shows the typical picture with fluid in both middle ears.

The cases of aero-otitis need no special mention, except that they occurred as a direct result of flying and apparently without infection of the nose or throat.

To complete the series, it might be added that in 19 cases swimming and diving were a predisposing cause, and that only 3 in the entire group went on to infection of the middle ear, developing an acute suppurative otitis media.

Our feeling concerning the management of any given case of acute catarrhal otitis media is that the exciting or predisposing cause should be determined if possible. If this is due to infection, as approximately 75 per cent of the cases in this series were, the infection should be eradicated first, and the treatment of the middle ear condition by the usual methods of inflation and removal of the serous fluid carried out. On the other hand, if no infection can be found in the nose or throat and cultures of the nose and nasopharynx are negative, some other cause must be looked for. First the problem of allergy must be considered since this factor seems to account for the greatest percentage of the "noninfectious group." If an allergy survey does not yield satisfactory results, then endocrine difficulties must be considered. Since most allergic individuals have

a consistently low basal metabolic rate, it is not advisable to order such a test on all patients of this group, lest we classify them all as hypothyroids. The basal metabolism plus a sugar tolerance test will differentiate those having a low rate due to allergy and those who really are hypothyroids.

In connection with this aspect of the noninfectious group we have been carrying on a systematic study of the problem for the past two years in the Research Laboratories of Syracuse University College of Medicine under a grant from the Hendricks Foundation. This work has not yet been completed and, in spite of many handicaps and disappointments due to the influence of the war, the work still goes on. We hope to publish our results at some later time, but the essence of our work is simply this: It is a comparison of the middle ear structures, microscopically, of normal and thyroidectomized pigs, and of pigs which have been sensitized to various allergens. We hope that this work will at some future date enable us to manage the treatment of cases of acute catarrhal otitis media on a sound scientific basis, rather than in the empirical fashion which is now in vogue.

In a recent paper on this subject, Robinson[1] suggested a classification of catarrhal otitis media and mastoiditis with effusion according to the presence or absence of coexisting disease in the nose, the sinuses or the nasopharynx.

He presents an excellent description of the serous effusion in the middle ear and states that "the effusion in acute catarrhal otitis media is an exudate; it has a high protein content with many polymorphonuclear leukocytes. The effusion in the subacute type of catarrhal otitis media is a transudate. There are few polymorphonuclear leukocytes, there is a low protein content, and when the fluid has been present in the tympanum long enough to cause the beginning of the chronic catarrhal stage with its adhesions, large mononuclear leukocytes and epithelial cells make their appearance."

Robinson's classification is a sound one, based on pathology. As for treatment, he advises the usual procedures, but also says that simple mastoidectomy should be done (as first suggested by Cody[2]) in catarrhal otitis media and mastoiditis with effusion when the more conservative measures have failed.

We feel that Robinson has given us a good workable classification of catarrhal otitis media when accompanied by infections

of the nose and throat, but that this should be broadened to include that important minority, the noninfectious group.

In conclusion let me say that it is not our intent to decry present methods of treatment. Rather, this paper was intended as an inventory of some of the facts concerning acute catarrhal otitis media and as a suggestion for classifying these cases on an etiological basis; namely, those due to infection and those due to some noninfectious cause. Other predisposing causes, as structural abnormalities of the nose and throat, should not be lost sight of. In the last analysis, this is a plea for the consideration of the patient as a whole and not as an isolated middle ear with eustachian tube attached. It is our opinion that only by such consideration can acute catarrhal otitis media be removed from the group of unsolved problems of otolaryngology.

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XXVIII

PREOPERATIVE TRAINING FOR DEVELOPMENT OF THE ESOPHAGEAL VOICE IN LARYNGECTOMIZED PATIENTS

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In no other organ of the body does carcinoma yield so successfully to early diagnosis and surgical treatment as in the larynx. Thomson,[17] in reviewing the history of carcinoma of the larynx, has said, "When we recall that 50 years ago Morell MacKenzie was obliged to say that for cancer of the larynx 'the only possible termination is death,' we may rejoice in realizing today that with laryngofissure we can essay lasting cures in over 80 per cent of the cases, that there should be no operative mortality, that surgery (laryngofissure and laryngectomy) will cure practically all cases of intrinsic cancer and that in skilled and careful hands they are now well established as safe and justifiable procedures."

The decision between laryngofissure and laryngectomy is not always easy, but if laryngeal surgeons would remember not to compromise with carcinoma, many more patients with cancer of the larynx would survive. A total laryngectomy is not a difficult operation technically, but if the surgeon is not willing to devote much time, effort and thought to these patients, to their social and psychologic problems, and to their rehabilitation, he should not deal with this type of case.

With the improvement in results of surgical treatment of cancer of the larynx, the major problems still to be solved in connection with this disease are education of the public regarding the curability of this type of cancer, and rehabilitation of the patient after laryngectomy. All authors who have written lately on the subject stress the fact that the majority of patients with cancer of the larynx do not consult a specialist in this field until the condition is far advanced. I regret that my own series of cases to be reported here show this same fact. The successful results following laryngectomy in early cases

Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, 1943.

and the deplorable failure of patients to seek treatment in time to avail themselves of its benefits prove that much remains to be done in educating the public to the fact that persistent hoarseness is a danger signal and should demand an immediate examination by a qualified specialist. Public health authorities, organizations devoted to the control of cancer, and the medical profession should combine forces to work toward this end.

When this is done, the problem of rehabilitation of laryngectomized patients will become increasingly important. The advancement of modern surgery has brought many refinements in the preoperative and postoperative care of the patient with cancer of the larynx. The laryngeal surgeon's responsibility should now extend beyond operative care and provide systematic training to insure a speaking voice after operation, for this is of prime importance to the patient. In the past, the greatest objection to total laryngectomy on the part of both surgeon and patient has been loss of the speaking voice. With preliminary voice training prior to operation this disability can be overcome.

In the series of cases in which I have performed laryngectomies, the problem of training the patient in the development of the esophageal voice has received special attention. The results of the method used have been so successful that I feel it should be more widely employed in the treatment of patients with carcinoma of the larynx.

THE ESOPHAGEAL VOICE

The esophageal voice has been widely studied, but from a review of the literature it is evident that the idea of preoperative training in preparation for its development is new. Heretofore, voice training has been delayed until after the larynx is removed. If a small part of this time and effort is devoted to training patients before operation, they can be assured of a speaking voice. Orton[13] says "every laryngectomized patient can talk with an esophageal voice if he makes up his mind to it." I agree with this, but the procedure is greatly simplified if training is begun before laryngectomy.

Czermak,[2] in 1859, described the first recorded case of esophageal voice. Störck[16] and Gersuny reported a case following total laryngectomy; Seiler[14] also reported an early case. A patient operated upon by Mikulicz was referred to Gottstein[3] for voice training, with brilliant results. Not only did the patient learn to speak, but his voice was capable of such modulation that he could sing within the range of almost an octave. In 1908, Gutzmann[4] reported 25 patients with intelligible voices following laryngectomy.

Morrison[8] has said: "It seems essential that all surgeons who perform total laryngectomies should attempt to aid these voiceless persons in securing a useful pseudovoice. Such patients will not be cut off in their social intercourse nor will they be unable to earn a living because of their lack of intelligible speech. In the postoperative period, the laryngectomized patient is apt to suffer from mental depression because of the social and economic barriers imposed on him by total aphonia. The best cure for this depression is a well-guided attempt to learn to produce the pseudovoice."

My interest in the esophageal voice was stimulated by one of the patients (Case 1) reported in this series. By sheer determination and practice he has developed an excellent speaking voice, which Stetson[15] has studied in detail.

Psychologic Preparation of the Patient. When a positive diagnosis of carcinoma following biopsy reveals the necessity for total laryngectomy, both patient and surgeon are anxious to carry out the procedure. The surgeon's concern over the outcome of the operation, for the time being, is likely to overshadow the thought of the patient's future life. This concern is commendable, but if the patient survives, the ability to converse with those around him is almost vital, so it is important to give some thought to his future, if he survives. If a few days' training before operation means the difference between a good speaking voice or the alternative of using some mechanical device, the patient is much better off if the operation is delayed until this training is given. The surgeon has only to compare the happiness of the laryngectomized patient with a pharyngeal voice to one bereft of speech to realize what this means. The training period is short—a week to ten days—and does not materially affect the chance of survival. The foremost thoughts in the mind of a patient about to undergo laryngectomy are: "Will I live?" and "Will I talk?" It is now possible to assure these patients that, if they live, they will talk.

Fortunate is the surgeon who has one or more patients who have developed a good esophageal voice. It is of inestimable value for the patient contemplating laryngectomy to see and talk to someone who has had the operation and has developed an esophageal voice. Those who have accomplished this inspire confidence, improve morale and stimulate hope in the one who is about to undergo this ordeal. Patients with an esophageal voice are far more successful in "selling" the operation than is the surgeon. One patient in this series said, "When I found out what I had and what should be done, I decided

to go home and use a gun, but after seeing and talking with C. L., I decided to have my throat cut instead."

Mechanics of Voice Production. "Voice is generally understood to refer to the production of sound by means of the pulmonary apparatus. There are many means by which an animal may throw the air in its neighborhood into sonorous vibrations either by voluntary or involuntary movements of some part of its body. In a wide sense voice may be defined as the purposive production of sound for purposes of communication. A narrower but more generally accepted view limits the sound to one made by means of the pulmonary air tract.[12]

"In developing phonation nature seems simply to have made use of the phylogenetically earliest primary valvular mud-excluding laryngeal function, using the opening and closing mechanisms in opposition to each other."[6]

Fluoroscopic studies made on my series of patients confirm the earlier observation made by Negus and Morrison that the narrowed cricopharyngeal space is the site of sound production after laryngectomy. The cricopharyngeal space makes the esophagoscopist's work more difficult, but it is a boon to the surgeon and patient after laryngectomy in the development of a pharyngeal voice. To students of this problem, the works of Negus, Morrison and Jackson on the cricopharyngeal space should be fundamental.

Preoperative Training. The surgeon should provide systematic training for the recovery of speech after laryngectomy. The esophageal voice is the result of timing and synchronization of movements of the lips, tongue and teeth, with adequate expulsion of air from the esophagus and pharynx. Definite narrowing of the cricopharyngeal space after laryngectomy creates sound vibrations when a sufficient volume of air is expelled from the esophagus. Therefore, development of an esophageal voice is dependent on the patient's ability to expel forcibly an adequate volume of air from the esophagus, that is, the act of belching. The laryngectomized patient's ability to develop an esophageal voice is absolutely dependent on his ability to master the technic of belching. If he does not learn this, he will not develop an esophageal voice. It is much easier to learn to belch properly before the larynx is removed than afterwards. If training is delayed until after operation, the glottic valve check to respiration is gone, and the patient will explode air from the trachea instead of the esophagus.

The belching effort is acquired easily by some patients; others find it quite difficult. By training patients for a week or ten days in this effort before laryngectomy, the greatest difficulty in the development of an esophageal voice is overcome. Most patients who must submit to laryngectomy desire a few days to arrange their affairs, and if this time be utilized in learning to belch, it will greatly shorten the postoperative training period for the development of the esophageal voice.

To aid these patients in learning to trap air in the esophagus, carbonated drinks are prescribed. The patient is told to inhale deeply, then cease active respiration while eructating air from the stomach and esophagus. The longer this is maintained, the greater the volume of air that will play up and down the esophagus. This procedure can be mastered with surprising speed.

Surgical Technic. The anesthetic used from 1936 to 1941 was avertin and local anesthesia. Since 1941, I have used sodium pentothal intravenously and local anesthesia. Sodium pentothal is superior to avertin, because the dosage is more easily controlled and the period of anesthesia is shortened, consequently decreasing the possibility of atelectasis and other pulmonary complications.

I have used the one-stage laryngectomy with subperichondrial dissection, as described by Crowe,[1] severing the superior cornu of the thyroid cartilage[18] and removing the larynx from below upward. Troublesome oozing is overcome by injecting the mucous membrane of the trachea at the site of amputation with one per cent novocain, and adrenalin (15 m. per ounce). A good closure prevents leakage, lessens danger of cellulitis and shortens convalescence.

After the larynx is removed and the pharynx is closed, the wound is filled with a mixture of two parts sulfathiazole and one part sulfanilamide. A Penrose drain and Dakin tubes are placed above and below, lateral to the incision, and the wound is irrigated through the Dakin tube with a five per cent sodium sulfathiazole solution every six hours. About the sixth or seventh day, if there is no apparent pharyngeal leakage, the patient receives water to swallow. If the pharyngeal opening has closed, the feeding tube is removed.

Postoperative Training. As soon as the feeding tube is removed, the patient is required to resume the belching effort. At first a great deal more stress is placed on having the patient belch than on having him try to form words. The words "O.K." and "Hello" seem to be the easiest to form. In the beginning, the patient is tempted to try to form sentences. This should be discouraged, for they should try

one word at a time. When this has been achieved, it is an easy matter to put phrases and sentences together.

The greater the cooperation and understanding between surgeon and patient in the postoperative training period, the better the results. The surgeon in directing this voice training should see the patient often, for there are many times when he and his family are discouraged. In my experience, though, few patients have been dilatory in attempting to develop an esophageal voice.

One should insist that the patient devote at least two or three hours a day to the effort to talk. If he will segregate himself from family and friends, he is more likely to keep at practicing than if someone is around. It is well to enlist the sympathy and cooperation of the wife in this undertaking for she can do much to encourage and reassure the patient, and to keep him practicing, when he might otherwise be negligent.

Clinical Results. The accompanying table shows details in 29 cases in which laryngectomy was performed in a series of 38 cases of carcinoma of the larynx seen at St. Luke's Hospital, Cleveland, Ohio, from 1936 to May, 1942. In addition to the 29 laryngectomies, 4 laryngofissures were performed. In the series subjected to laryngectomy, 13 were intrinsic growths and 16 were extrinsic. Several of the intrinsic growths were far advanced.

The great proportion of late cases in this series emphasizes the need for education of both the profession and the public in recognition of the early signs and symptoms of cancer of the larynx.

Of the 29 patients, 19 had no preliminary training for development of the esophageal voice, and only 3 in this group developed a good speaking voice. The remaining 10 patients had preliminary voice training and all developed a good voice following laryngectomy. The ability to be understood over the telephone has been set as an arbitrary standard in judging the voice.

SUMMARY

A series of 29 laryngectomies performed in a series of 38 cases of carcinoma of the larynx treated at St. Luke's Hospital, Cleveland, Ohio, during the period from 1936 to 1942 illustrates the value of preliminary voice training for laryngectomized patients. Of 19 who had no preliminary training in the development of the esophageal voice, only 3 were able to develop the ability to speak following operation. Of the 10 patients who had preliminary training, all developed

the ability to speak well enough to be heard over the telephone, and several had excellent voices.

Patients to be subjected to total laryngectomy should have voice training before, not after, operation because (1) development of a pharyngeal voice is dependent on the patient's ability to belch; (2) it is much easier to learn to belch before the larynx is removed than afterwards; and (3) the time required to master the technic of belching is short (one week to ten days) and does not jeopardize the patient's chance of survival.

The proportion of advanced cases of laryngeal cancer in this series is appallingly high, as in other reported series. This shows the necessity for educational and public health measures to combat this disease. The great improvements in the technic and the management of these cases have not resulted in corresponding improvement in general results, because so many patients do not avail themselves of treatment until the lesion is far advanced.

610 ROSE BUILDING.

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TABLE 1.—SHOWING DETAILS OF 29 CASES IN WHICH LARYNGECTOMY WAS PERFORMED

Case	Age	Sex	Occupation	Pathology	Operation	Voice Training	Results
1.	48	M	Business executive	Late intrinsic carcinoma, I	Laryngofissure. Total laryngectomy 1 month later	No	Excellent speaking voice. Can also sing
2.	53	M	Business executive	Carcinoma, II, right cord. Extensive leukoplakia, both cords	Total laryngectomy	No	Good voice
3.	59	M	School superintendent	Carcinoma, I, anterior third of right cord and commissure	Total laryngectomy	No	Good voice
4.	62	M	Owner shoe store	Carcinoma, III, anterior two thirds of right cord and commissure	Total laryngectomy	No	No voice. Alive and well
5.	66	M	Mechanic	Carcinoma, II, left cord and commissure; cervical metastasis	Total laryngectomy	No	No voice. Recurrence and death 1 year after operation
6.	55	M	Laborer	Carcinoma, III. Fungating mass entire right side of larynx	Total laryngectomy	No	No voice. Recurrence and death 1 year after operation
7.	53	M	Laborer	Carcinoma, III	Total laryngectomy	No	No voice. Recurrence and death 7 months after operation

8.	45	M	Trolley motorman	Carcinoma, IV, ulcerating and perilaryngeal abscess	Total laryngectomy	No	No voice. Recurrence and death 8 months after operation
9.	45	M	Saloon keeper	Carcinoma, III, right true ventricle and false cord with subglottic extension	Total laryngectomy	No	No voice. Recurrence and death 4 months after operation
10.	53	M	Farmer	Carcinoma, II. Fungating ulcerative mass, right cord. Extension to epiglottis	Total laryngectomy	No	No voice. Recurrence and death 7 months after operation
11.	67	M	Farmer	Carcinoma, III. Perilaryngeal extension. Erosion of thyroid cartilage with abscess. (Refused operation 6 months earlier)	Total laryngectomy	No	No voice. Recurrence and death 8 months after operation
12.	54	M	Machinist	Carcinoma, II, both cords and ventricles. Extension to esophagus	Total laryngectomy	No	No voice. Alive and working
13.	54	M	Mechanic	Carcinoma, III, extrinsic, with metastases to cervical glands	Total laryngectomy. Removal of right sternomastoid muscle. Ligation of right internal jugular vein	No	No voice. Died 3 months later from hemorrhage

TABLE 1 (CONTINUED)

CASE	AGE	SEX	OCCUPATION	PATHOLOGY	OPERATION	VOICE TRAINING	RESULTS
14.	52	M	Railroad section hand	Carcinoma, III, left cord and commissure	Total laryngectomy	No	No voice. Alive and well
15.	60	M	Watchman	Carcinoma, III, posterior larynx and lateral wall of pharynx	Total laryngectomy	No	No voice. Recurrence and death 6 months after operation
16.	53	F	Housewife	Carcinoma, II, left cord and commissure	Total laryngectomy	No	No voice. Alive and well
17.	66	M	Laborer	Carcinoma, II, posterior larynx and pharynx	Total laryngectomy	No	No voice. Died 8 months after operation
18.	76	M	Painter	Carcinoma, III, epiglottis. Extension down larynx and lateral wall of pharynx	Total laryngectomy	No	No voice. Died 1 month later of heart disease
19.	64	M	Attorney	Carcinoma, II, left cord and commissure, half of right cord	Total laryngectomy	1 week	Good voice. Continues active law practice
20.	57	M	Baker	Carcinoma, II, base of epiglottis, extending to larynx	Total laryngectomy and removal of epiglottis	2 days	Fair speaking voice. Well

21.	40	M	Manager telephone company	Carcinoma, I, right cord and commissure, anterior left cord	Total laryngectomy	1 week	Very good voice. continues work
22.	63	M	Civil engineer	Carcinoma, I, posterior two thirds of right cord; arytenoid fixation	Total laryngectomy	2 days	Good voice
23.	45	M	Storekeeper	Carcinoma, III. (Refused operation 8 months earlier)	Total laryngectomy	1 week	Excellent voice 30 days after operation. Voice heard distinctly in large assembly hall. Recurrence and death 14 months later
24.	52	M	Painter	Carcinoma, III, left cord, ventricle and commissure	Total laryngectomy	1 week	Fair voice, improving rapidly
25.	52	M	Mechanic	Carcinoma, III, entire larynx with erosion of thyroid cartilage and right lobe of thyroid. (Operation refused 1 year earlier)	Total laryngectomy and right thyroidectomy	3 days	Fair voice. Improving rapidly. No evidence of recurrence
26.	60	M	Plumber	Carcinoma, III, entire right cord, ventricle, commissure and anterior left cord	Total laryngectomy	1 week	Fair voice; improving rapidly.
27.	48	M		Chondroma, larynx with invasion both thyroid lobes. Previous attempt elsewhere to do laryngectomy abandoned because of hemorrhage	Total laryngectomy and ligation of right external carotid. Skin graft required	No	No voice. Alive and well 18 months after operation

TABLE 1 (CONTINUED)

CASE	AGE	SEX	OCCUPATION	PATHOLOGY	OPERATION	VOICE TRAINING	RESULTS
28.	61	F	Housewife	Carcinoma involving right cord, commissure, anterior left cord	Total laryngectomy	5 days	Good speaking voice 10 days after laryngectomy
29.	52	M	Painter	Carcinoma, left cord, ventricle and commissure	Total laryngectomy	10 days	Good speaking voice 6 days after laryngectomy

XXIX

PRIMARY CARCINOMA OF THE EUSTACHIAN TUBE

A STUDY OF THE EVIDENCE OF ITS OCCURRENCE

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EVANSTON, ILL.

Occasional reports of primary carcinoma of the eustachian tube appear in the literature. These primary carcinomas are to be distinguished from secondary involvements by tumors originating in other nasopharyngeal locations. The lesions should ordinarily be identified early because there have been excellent studies and reports designed to encourage more accurate and more frequent examinations of the nasopharynx. There are still too few accurate diagnoses made while the patient is alive and too many lesions seen in the advanced stages either because they are overlooked or because the patients ignore their early symptoms and come for examination after the symptoms are well established. It is the purpose of this paper to review the clinical features and the literature, to report a case and to make some comments on therapy. These reports again emphasize the advisability of searching the nasopharyngeal approach to the ear when there is persistent eustachian tube obstruction. When cervical metastases appear and the primary lesion is not evident, the possibility of eustachian tube tumor should not be overlooked. One pathologist[18] has stated that at present the only circumstance in which a diagnosis of carcinoma of the eustachian tube can confidently be made is the one in which a complete necropsy has established the absence of all primary neoplasms in the craniocervical region and in all other parts of the body likely to yield metastatic deposits in the cervical lymph nodes, and in which the eustachian tube lesion has been positively identified. The alert examiner will recognize and make a biopsy of the first tumor bulge associated with persistent tubal closure, especially one which prevents inflation. If the patient fails to report at this time, he will later present himself for examination with typical symptoms of lateral wall nasopharyngeal tumor which are: (1) pain due to irritation of the first and second branches of the trigeminal nerve, (2) postnasal bleeding, (3) obstruction to nasal

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Read before the Chicago Laryngological and Otological Society, January 4, 1943.

breathing, (4) obstruction of the eustachian tube with tinnitus and deafness, and frequently these are associated with (5) painless unilateral cervical adenopathy.

From cases reported it seems highly probable that a fair proportion of lateral nasopharyngeal tumors originate in one tube. Salinger and Pearlman[11] in an important study of the pathologic classification of nasopharyngeal tumors noted that in the majority of their cases (60 per cent) the mass originated in the lateral wall in the region of the eustachian tube or Rosenmüller's fossa. Textbooks and monographs give little or no mention of primary eustachian tube carcinoma nor are they specific as to the exact origin of tumors in this region. Frequently, too little evidence is presented to prove the eustachian tube origin of the carcinoma. Judging from published reports primary carcinoma of the eustachian tube is only rarely demonstrated. Secondary involvements and metastatic temporal bone changes are more frequently reported. The appearance of symptoms referable to any of the cranial nerves other than the first and second divisions of the trigeminal nerve is an indication of extension of the tumor through the basal foramina, the jugular foramen, or the orbital fissure and introduces the hopeless stage.

Negri[8] in 1888 reported a patient who was observed for four years and whose condition was correctly diagnosed. A syndrome was observed of unilateral headache, tinnitus, trigeminal neuralgia, paralysis of the oculomotor nerve, ptosis and exophthalmos. Autopsy revealed intracranial invasion. There was an irregular tumor mass with ulceration in the orifice of the tube and tumor tissue invading the fossa of Rosenmüller.

Proctor[4] in 1912 reported a patient who complained of persistent tubal obstruction. One tube was occluded by soft granulation tissue which bled easily. The lymph nodes of that side were enlarged. The tumor grew rapidly. Biopsy revealed a highly malignant carcinoma. The patient died seven months after examination, apparently from cerebral involvement through the jugular foramen.

Jacod[5] reported the following cases with diagnoses of sarcoma. They are included here because I believe that they would probably now be reclassified and most of them called transitional cell carcinomas.

1. (1915) A man, aged 68, complained of deafness and tinnitus in the left ear, pain in the temporal region, and loss of appetite. The tubal mucous membrane projected from the cavity of the tube and a firm circumscribed tumor was palpable, covered by thickened mucosa

without ulceration. The mass gradually increased to occupy all of the lateral wall of the nasopharynx. Five weeks after the known onset the superior maxilla was resected and an ovoid tumor was detached from the base of the skull. It appeared to rise from the mucous membrane in the cartilaginous portion of the tube. One month later a chain of enlarged lymph nodes was removed from the neck. The patient died five months after the known onset. Autopsy revealed a tumor infiltrating the entire cartilaginous portion of the tube.

2. *(1915)* A man, aged 53, complained of deafness and tinnitus in the left ear, obstruction of the left nostril, pain in the left ear, the left side of the nasopharynx, and diffusely over the left side of the head. Examination of the nasopharynx revealed deformity and induration at the orifice of the left eustachian tube. This gradually enlarged. The left superior maxilla was resected and the tumor removed. There was recurrence in two months with regional lymph nodes. The patient died six months after the known onset and three months after the operation. There was no autopsy.

3. *(1921)* A man, aged 64, complained of deafness and tinnitus in the left ear, trigeminal neuralgia, obstructed left nostril, paralysis of the trochlear and abducens nerves, and almost complete loss of function of the oculomotor nerve on the left side. The left eustachian tube appeared tripled in size. There was a firm tumor in the region of the tube which on biopsy was diagnosed a small round-cell sarcoma. Seventy-eight milligrams of radium were used for 3 hours and again for 22 hours. All symptoms improved. The patient died six months after the known onset and one and a half months after the first radium treatment.

4. *(1921)* A man, aged 32, suffered from trigeminal pain, headache, restricted motility of the left eye, reduced auditory acuity, and tinnitus. There was a tumor of the left wall of the nasopharynx. The patient died seven months after the known onset of the disease. At necropsy a large tumor, diagnosed as a sarcoma, was found involving the eustachian tube, the lateral wall of the nasopharynx, and extending to the left anterior cerebral fossa.

5. *(1936)* A man, aged 40, complained of left otalgia with intermittent left temporal headache. Upon examination the eustachian catheter met with obstruction and was bloody. The tube was tumefied at the level of the anterior cushion. The thickening extended to the fossa of Rosenmüller. There was no enlargement of the cervical glands. The biopsy specimen was diagnosed reticulum-cell sarcoma. Twenty-two international units of x-ray were given over a

three-week period. Three months later an operation by the transmaxillary approach was made with resection of the tube. Subsequent data is not available.

Charleux and others[7] in 1935 reported a man, aged 60, who complained of pain in the left ear, in the side of the face, and in the left temperoparietal region. There was deep soreness on breathing. Later there was paralysis of the left oculomotor and abducens nerves with diplopia. The tongue deviated to the left. There was a tumor in the region of the left eustachian tube and salpingoscopy revealed a reddish growth in the region of the pavilion of the tube. The biopsy diagnosis was prickle-cell epithelioma. Radium relieved the pain but did not modify the tumor. No further record is available.

Dunlap[13] in 1938 reported observations of 16 cases of this disease in six years, all in Chinese. There were present as symptoms: deafness, hemorrhage, and nasal obstruction. Metastases were noted in most cases. Biopsies all revealed low-grade transitional-cell carcinomas which appeared to be radiosensitive.

Stewart and Lieber[18] in 1940 reported a man, aged 50, who had a lump on the left side of the neck, constant left-sided headache, earache, and a painless, freely movable mass beneath the left mandible for one year. A preliminary diagnosis was made of Hodgkin's disease or lymphadenitis. There was a bulging inward of the left pharyngeal wall at the level of the tube. Cervical gland biopsy revealed squamous-cell carcinoma, probably metastatic. The patient died 26 months later. There were 2360 roentgens of x-ray given. Autopsy revealed primary squamous-cell carcinoma in the left eustachian tube. There was extension in the connective tissue around the left internal carotid artery and internal jugular vein into the base of the skull and cranial cavity with metastases to the cervical lymph nodes .

Schumaker[17] in 1940 reported a cure in one patient and an uncertain outcome in another, and counselled that the definite clinical picture and the pathologic findings, practically duplicated in the two cases, should bring earlier diagnoses in subsequent cases.

1. A 54-year-old Austrian woman complained of left-sided deafness for two months. The left drum was yellow and revealed a fluid level. Bi-weekly inflations had been given with no improvement in the symptoms. The record of the examination at the first visit made specific mention of normal eustachian orifices. The first appearance of the tumor was as a round, red, cauliflower-like growth issuing from the left eustachian tube, movable as if attached by a pedicle, and of an approximate diameter of 12 mm. The biopsy

diagnosis was highly malignant squamous epithelioma. Treatment was by radium. Contact with the patient was lost and the ultimate result was not known.

2. A 60-year-old American man complained of deafness with a feeling of fullness in the left ear of six weeks' duration despite repeated inflations. The tympanic membrane was yellow and revealed a fluid level. Inflation of the left eustachian tube was accomplished with unusual difficulty and without relief. On the thirteenth week after the onset of symptoms a minute, red, cauliflower growth was observed in the atrium of the left eustachian tube. Biopsy was possible 16 weeks after the onset of symptoms. The report was grade 4 squamous-cell epithelioma. No cervical glands were palpable. One 2.1 millicurie gold radon seed was buried deep in the center of the tumor. Also 300 units of deep x-ray were given over the left side of the neck, repeated three times at weekly intervals. On the fifty-first day after radon application there was no evidence of the tumor. The left eustachian orifice and the drum appeared normal. Monthly observations were carried out until three years later when the patient died of a coronary accident. The region of the left eustachian tube was studied and all structures, including the tube and its atrium, were macroscopically and microscopically free from tumor cells.

Brownell[16] in 1940 found six pertinent cases in the foreign literature but none in the English or American literature. He reported two cases on Furstenberg's service at the University of Michigan. One was suspected and proved at autopsy, the other was unsuspected and demonstrated at autopsy.

1. A 33-year-old man noticed deafness and tinnitus in the right ear with associated right occipital headache. There was a swelling just below the right mastoid tip. The right drum membrane was dull and the landmarks were poor. Mirror examination of the nasopharynx was not accomplished. Cervical adenopathy was present. Biopsy revealed a poorly differentiated squamous-cell carcinoma. There were 5800 roentgens of x-ray given over the right cervical region in fractional doses over a period of one month. At postmortem examination a nodular mass 3 cm. in diameter was found in relation to the midportion of the right eustachian tube. The mucous membrane of the nasopharynx was not involved. Metastases were present in the ribs, sternum, vertebrae, cranium, spleen and liver.

2. A 57-year-old man developed intermittent then constant pain in the left ear. There had been no previous disease and no otorrhea. Pain developed behind the left eye, followed soon by diplopia. The

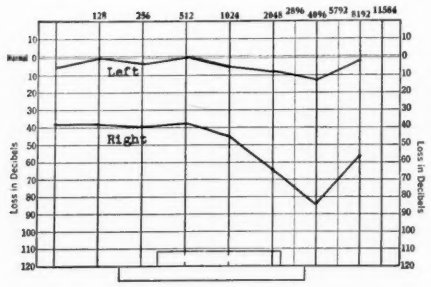

Fig. 1.—Audiogram made at the first examination.

left tympanic membrane was red and thick and a polyp projected through a posterior perforation. There was profound deafness, abducens palsy, and hyperesthesia over the left trigeminal nerve distribution. A Gradenigo syndrome developed and a diagnosis was made of probable petrositis. The patient expired. Autopsy revealed that a medullary squamous-cell carcinoma had completely replaced the outer third of the left eustachian tube, extending widely to infiltrate the petrosa, the infratemporal fossa, and the gasserian ganglion.

REPORT OF THE AUTHOR'S CASE

A man, aged 59, came for examination because of slight bleeding from the throat. Examination of the pharynx failed to reveal a source of the hemorrhage. Examination of the nasopharynx revealed a tumor mass apparently originating in, or at the region of, the right eustachian tube, and already partly obstructing the right nostril. The history points definitely to the eustachian tube origin, for the tube had been blocked for many months, and a consulting otologist had been unable to inflate the right ear. A careful search did not reveal enlarged cervical glands. The patient was very active, had lost no weight, and complained chiefly of deafness in the right ear, tinnitus, partial blockage of the right nostril, and slight recent bleeding. Biopsy revealed transitional-cell carcinoma. Early examination was delayed because of pressing duties and was then made only because of the annoyance of bleeding.

Intensive x-ray treatment was given over the tumor. There was external radiation through two ports amounting to a total of

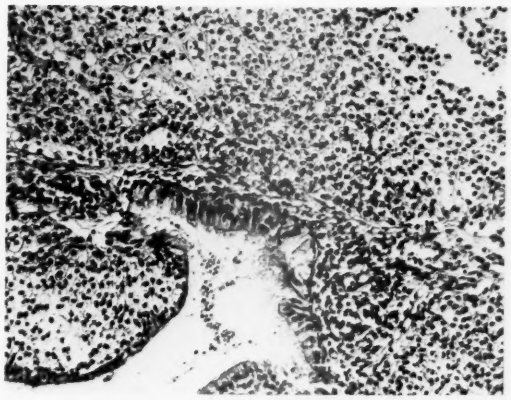

Fig. 2.—Photomicrograph of specimen taken for biopsy.

10,000 roentgens, to the lateral right nasopharynx 5150 roentgens, and to the anterior right malar region 4450 roentgens. Three right upper teeth were removed to facilitate the treatment. The factors were 400 kv., 3 mm. copper filtration at 75 and 80 cm. distance. Following these treatments there was marked regression of the tumor, although it was still palpable and visible. It was then decided to implant radon seeds in the tumor. Six 2-millicurie seeds were implanted. This was followed by a rather intense reaction with dryness of the throat, voice disturbance and some general weakness.

AGE AND SEX INCIDENCE

In this series of 14 cases, 13 men, ages 32 to 68, were reported and one woman, aged 54. Few reports of nasopharyngeal tumors give the exact location at the first examination. Twelve of Salinger and Pearlman's 24 cases gave a definite record of the eustachian tube or the fossa of Rosenmüller as the place of origin. Pathologists rarely explore the region of the eustachian tube. However it can be rather easily done with a mirror. In malignant cervical metastases not otherwise explained, the eustachian tube location must be suspected in the search for the original focus. The majority of reports simply state that a neoplasm was found about the orifice of the eustachian tube or in the fossa of Rosenmüller, with involvement of the former.

TABLE 1—PRIMARY CARCINOMA OF THE EUSTACHIAN TUBE

EVIDENCE FROM THE LITERATURE

AUTHOR	LIFE AFTER KNOWN ONSET	YEAR REPORTED	TREATMENT	PATHOLOGY	RESULT	CAUSE OF DEATH	CERVICAL GLANDS
Negri	4 years	1888	Supportive	Carcinoma	Died	Intracranial invasion	Not reported
Proctor	7 months	1912	None	Carcinoma	Died	Cerebral involvement through jugular foramen	Yes
Jacod	5 months	1915	Superior maxilla resection	Small round cell sarcoma	Died	Invasion through base of skull	Yes
.....	6 months	1915	Superior maxilla resection	Small round cell sarcoma	Died	Invasion through base of skull	Yes
.....	6 months	1921	Radium and surgery	Sarcoma (?)	Died	Invasion through base of skull	Not reported
.....	7 months	1921	Surgery	Sarcoma (?)	Died	Extension to anterior cerebral fossa	Not reported
.....	Unknown	1936	X-ray; funnel resection of tube	Reticulum cell sarcoma (?)	Unknown	No data available	Not reported

TABLE 1 (CONTINUED)

AUTHOR	LIFE AFTER KNOWN ONSET	YEAR REPORTED	TREATMENT	PATHOLOGY	RESULT	CAUSE OF DEATH	CERVICAL GLANDS
Charleux et al	Unknown	1935	Radium	Prickle cell epithelioma	Unknown	No data available	Not reported
Dunlap	Series of 16	1935	Radium	Carcinoma	Died	Usually intracranial invasion	50%
Stewart, Lieber	3 years	1940	X-ray and Manganese injections	Squamous cell carcinoma	Died	Intracranial invasion	Yes
Schumaker	Unknown	1940	Radium	Squamous cell carcinoma	Unknown	Unknown	None
.....	3 years Coronary	1940	Deep x-ray; radium	Squamous cell carcinoma	Cured	.....	None
Brownell	14 months	1940	Deep x-ray	Squamous cell carcinoma	Died	Distant metastases	Yes
.....	16 months	1940	Deep x-ray	Squamous cell carcinoma	Died	Intracranial extension	Not reported
Lawson	Living	1942	Deep x-ray; radon	Transitional cell carcinoma	Living	.....	None

SYMPTOMS

The symptoms of primary carcinoma of the eustachian tube are:

1. Early eustachian tube obstruction; tinnitus; deafness.

2. Pain due to irritation of the first and second branches of the trigeminal nerve.

3. Postnasal bleeding when the tumor becomes vascular.

4. Some degree of unilateral nasal obstruction.

5. Cervical adenopathy, unilateral and painless; present in 50 per cent of the cases.

6. Late symptoms referable to cranial nerves other than the first and second divisions of the trigeminal nerve. These indicate hopeless extension through: the basal foramina, the orbital fissure, or the jugular foramen.

PATHOLOGY

Biopsy specimens from the eustachian tube were taken in 6 cases in the series. One case revealed at operation a firm tumor mass attached to the base of the cranium and arising from the cartilaginous portion of the tube. Necropsy findings were reported in cases of Negri, Jacod (case 2, 1921), Brownell, Schumaker, and Stewart and Lieber. It is apparent in most instances that the neoplasm arose from the cartilaginous portion of the tube. In one case there was a firm small tumor attached to the base of the skull in the temporosphenoidal region with early extension into the middle and anterior cerebral fossae through the jugular foramen and the sphenoidal suture. The cranial nerve trunks at this level when compressed give resultant paralysis, especially those of the trigeminus and abducens and occasionally the optic, oculomotor, trochlear nerves. Extension of the growth to the sphenoidal fissure and the orbit was responsible for the development of the ocular syndrome in some cases. Brownell's second case revealed extensive destruction of the base of the skull without invasion of the nasopharynx. In Stewart and Lieber's patient the mucosa of the nasopharynx was not invaded. Enlargement of the cervical lymph nodes was present in only five cases. Early metastatic involvement of these nodes was present in at least three. There was only one record of distant metastases.

Jacod diagnosed five specimens as sarcoma, four as small round cell sarcoma, one as reticulum sarcoma. Negri, Proctor, and Brownell simply gave the diagnosis of carcinoma. Charleux and his asso-

ciates reported prickle-cell epithelioma. Stewart and Lieber reported squamous-cell carcinoma. Salinger and Pearlman concluded that the diagnoses were confusing but that most of them were transitional-cell carcinomas or lymphoepitheliomas.

CLINICAL FEATURES

There is fullness in the ear and intractable pain as the disease progresses. Headache may be temporal or trigeminal and it increases in severity and constancy.[1] In this series headache was associated with pain in the ear in 4 cases followed by nasal, retronasal, or nasopharyngeal pain in 3 cases. Pain in the neck was present late in 2 cases. There were symptoms referable to the ear in all cases. There was diminished acuity of hearing and tinnitus. Auditory symptoms alone ushered in the disease in 5 cases. There were symptoms referable to the eye in 4 cases: reduced visual acuity, diplopia, ophthalmoplegia, and in 1 case early blindness. There was evidence of partial or complete paralysis of the trochlear and abducens nerves in all patients with eye involvement. There was epistaxis of slight amount in 3, and nasal obstruction in 3.

PHYSICAL FINDINGS

There was early evidence of tumor involvement of the eustachian tube in 9 cases. In Brownell's second case repeated examinations gave negative results. A firm mobile tumor at the level of the tubular eminence was detected in 7 cases, usually with inward or forward bulging of the pharyngeal wall at this level. Ulceration was not a prominent feature. Salpingoscopy gave evidence of complete obstruction in 6 cases. The drum was retracted, and there was slight redness, like ordinary tubotympanitis, with or without effusion.

TREATMENT

There was surgical treatment in 5 cases. In Proctor's case the glands of the neck were excised because of pain in that region. The patient was relieved of the pain but died seven months after the onset of the symptoms. In Jacod's case 1, 1915, the superior maxilla was resected and an early detached tumor removed from the base of the skull. One month later lymph nodes were removed from the neck. There was recurrence of the growth in the nasopharynx, and the patient died five months after the onset. In Jacod's case 2, 1915, there was resection of the superior maxilla, recurrence of the tumor with involved lymph nodes, and the patient died six months after the

onset. In Jacod's 1936 case there was a transverse maxillary incision, resection of the posterior portion of the nasal septum, and a funnel-shaped resection of the eustachian tube in its cartilaginous portion. The ultimate fate is unknown. In this case x-ray therapy was used two months before the operation, a total of 2000 i. u. being given. X-ray or radium was used in 11 cases. In Jacod's case 1, 1921, two tubes containing 78 mg. radium were placed in the left nostril, one application for 3 hours and one for 22 hours. This was followed by relief of pain, reduction in the size of the tumor, ability to breathe through the nostril, improved hearing, and finally no evidence of the tumor. The patient died six months after the known onset.

The patient of Stewart and Lieber received intensive x-ray therapy followed by intravenous chemotherapy with lead and manganese solutions. Pain and swelling disappeared. Four months later there was a recurrence. A second course of x-ray treatment was given. The patient died two years after the first course of x-ray treatment and three years after the onset of the condition.

Whether x-ray or radium element is employed, the underlying principles outlined by Coutard should be given proper consideration. One method suggested is to have the primary lesion and the lympathic region involved exposed daily or twice daily to x-ray or radium over periods varying from 20 to 60 days. Adequate filtration is used to obtain the most highly penetrating rays. The total dose administered is estimated on a biologic basis, the aim being to cause complete regression of the growth without permanent damage to the surrounding normal structures. When the primary lesion is small and localized, surface applications of radium or radium and radon are combined with external x-ray therapy.

COMMENT

Neoplasms of the eustachian tube should be suspected in the presence of persistent obstruction of the tube and in the absence of evident nasopharyngeal lesion to account for metastatic cervical lymph nodes, especially where there is head and ear pain not otherwise accounted for. Few clinical reports are sufficiently complete to absolutely identify the eustachian tube origin of the tumors.

The patient mentioned in my case report is not presented as a cure. He has responded well to treatment. The tumor mass has disappeared. There are no symptoms of extension nine months after the tumor was discovered and he is carrying on full time in his usual occupation.

CONCLUSIONS

The nasopharynx should be painstakingly and repeatedly searched in the presence of persisting eustachian tube blockage.

The most favorable treatment would appear to be deep x-ray therapy combined with radium or radon.

There is a tendency of these tumors to make regional invasion, particularly into the skull, even though the nasopharyngeal tumor mass may seem to thoroughly regress.

The early symptoms are not disabling. Therefore, when patients are seen late effective control of the neoplasms cannot be achieved.

More recent cases treated with radiation according to the Coutard principles appear to survive longer.

The results with accurate placement of radon would seem to warrant further clinical trial.

636 CHURCH STREET.

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XXX

A PHARYNGEAL SYNDROME PROBABLY OF VIRUS ORIGIN

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During the past 18 years I have observed and kept records of 17 cases of a syndrome consisting of herpetic lesions in the pharynx and mouth, accompanied by pain in the ear and the occipital regions and an area of paresthesia in the parietal region. The occurrence of 20 additional cases of this syndrome during a period of six weeks, during which we had an epidemic of keratoconjunctivitis, suggested a possible virus origin of this syndrome and prompted me to make this preliminary report.

While there were some variations in the group seen previous to the epidemic, the two groups were clinically similar. The patient's chief complaint was the pain in the ear which was of a sharp, shooting type, distinctly neuralgic in character and never lasting over one minute. The pain was aggravated by an increase in intracranial pressure produced by coughing. The drum membrane and the canal appeared normal. There was no diminution in hearing. The area of paresthesia over the parietal region is typical in these cases and covers an area corresponding approximately to the palm of the hand. There was usually no pain over this area, the hair simply felt as if it were crossed or brushed down the wrong way.

The pharyngeal lesions varied from a single ulcer on the buccal membrane to a massive area of herpetic lesions covering the entire soft palate, both pillars and the posterior pharyngeal wall. The lesions were always unilateral. The bases of these areas formed pseudomembranes which were hemorrhagic on removal of the membrane. The areas themselves were not particularly painful, but were extremely sensitive to salt and fruit juices.

Seven of the 20 patients in the epidemic group complained of severe pain in the occipital region on the affected side. This was not noticed in the previous cases.

Read before the Western Section meeting of the American Laryngological, Rhinological & Otological Society, Portland, Ore., January 31, 1943.

Cultures from the throat were taken in all cases and no pathogenic organisms were found. The usual report was the finding of an occasional staphylococcus. The outstanding finding from a laboratory standpoint was the predominence of lymphocytes in scrapings from the base of the ulcer and from fluid aspirated from unruptured herpetic blebs[1]. The blood count, the hemoglobin and serologic tests seem of no significance.

Ten of the 20 patients in the epidemic group also suffered from keratoconjunctivitis and in these cases the pharyngeal syndrome seemed less severe. The adenopathy characteristic of the keratoconjunctivitis was not noticed in the group where lesions were limited to the pharynx, but the preauricular node was enlarged in all cases with eye involvement.

The cases seen several years ago were considered of focal origin, and infected teeth and tonsils were removed. Later vitamin deficiencies took the leading place as a causative factor and, even after reviewing the work of Goodpasture[2] in relation to herpes zoster and virus infections in 1931, the possibility of a virus origin was not given serious consideration in this syndrome until the recent outbreak of keratoconjunctivitis.

Where contacts could be traced, the incubation period was from five to seven days. There were no general symptoms; the temperature rarely rose above 100° F. The ages of those afflicted ranged from 22 years to 56 years.

That the condition was highly contagious was demonstrated by the fact that up to four cases appeared in one family, some members having the pharyngeal syndrome, some the typical keratoconjunctivitis, and others both. The average duration was 12 days.

The treatment was palliative. It consisted of acetophenetidin and acid acetyl salicylici with codein phosphate for pain, mild alkaline irrigations plus the application of dye antiseptics to the pharyngeal lesions, and large doses of vitamin B_1 for its antineuritic action. Sulfonamide compounds were used but they did not affect the course of the disease.

The neuritis, particularly that involving the posterior occipital region, was of longest duration, some patients still having occasional attacks of pain four months after the onset.

CONCLUSIONS

1. A pharyngeal syndrome suggesting a virus infection is reported.

2. An apparent immunity seems to be rapidly established.

3. Since this is a preliminary report certain details, such as the definite anatomical basis, have not been completed.

4. No specific treatment for the condition was found.

2730 COLBY AVENUE.

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XXXI

CHEMOTHERAPY IN NOSE AND THROAT DISEASES

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The status of the sulfonamide drugs is changing so rapidly that new and improved combinations are being produced and tried continuously. It is evident that the rapid changes are creating a demand for frequent reports of progress. This paper has been prepared in an effort to supply this demand.

It is now about six years since the introduction of the sulfonamide group of drugs to the realm of medical and surgical practice. During this period the various types of sulfonamide drugs have been accepted, studied, and to a certain degree correlated not only in their general action but furthermore in their action in the special fields of practice. Thus, it would not seem amiss at the present time to attempt, in the specialty of rhinolaryngology, an evaluation of this group of extremely important medicaments, both as a group and as individuals.

In the years 1936 and 1937, sulfanilamide was almost alone in representing this group, and it is not necessary to say that we knew very little of its actual mode of action, dosage, and toxicity, nor had we any actual scientific knowledge to ascertain why, in certain cases of infection, we were apparently obtaining such unusual and even spectacular results. The entire administration of the drug was not as we see it now, but based on a hit or miss method, hoping that our good results would more than counteract our complications and bad results. As time passed, other members of a group appeared, each succeeding drug being more efficacious and less toxic than its predecessor. With our improved laboratory technique we were able to follow very carefully the blood concentration and to determine more accurately the possible complications and their gravity, or lack of importance. This, in brief, is the history of the development of the so-called sulfonamide drugs.

From the Manhattan Eye, Ear and Throat Hospital, New York City.

Read before the Sixty-fourth Annual Meeting of the American Laryngological Association, Atlantic City, N. J., May 25, 1942.

Sulfanilamide, the first one of the sulfonamide group to be employed as a chemotherapeutic agent, was not the result of one experiment that happened overnight, but was the result of over 25 years' experimental work by such great men as Gelmo, Horlein, Dressel, Sisley, Domagk, Long, and many others. Gelmo, in 1908, was the first to synthesize sulfanilamide while working with the azo-dyes, and he gave a very accurate chemical description of it, but did not apply it experimentally or clinically. Little by little, all of these scientists added to the work of their predecessors, until 1932 when Domagk noted the potency of certain dyes in combatting streptococcus septicemia in mice. By adding an azonaphthalene ring and including the sulfonamide group, a new drug was synthesized, namely, prontosil. He therefore deserves the credit for having been the first to use the sulfonamide group in animal experiments, and it was he who first suggested the clinical application of the same. For this work he was awarded the Nobel Prize in Medicine in 1939.

Since 1937, numerous drugs of this group have been synthesized, and, in general, each succeeding one seemed to act better and with less toxicity than the last. So many of these drugs have been produced in the past five years that one must be on the alert to keep up with the advance in this particular phase of chemistry. Since sulfanilamide was first brought out, no less than 18 of these drugs have been introduced to the medical profession, namely: prontosil, neo-prontosil, prontylin, sulfapyridine, sodium sulfapyridine, sulfathiazole, sulfamethylthiazole, sodium sulfamethylthiazole, sulfadiazine, sodium sulfadiazine, sulfaguanidine, dodecanol sulfanilamide, sulfapyrazine, sodium sulfapyrazine, promin (para-diamino-diphenyl sulfone), sulfacetimide, succinyl sulfathiazole.

In the beginning, some of these drugs were heralded as being specific for a particular organism: sulfanilamide for hemolytic streptococci, sulfapyridine for pneumococci and sulfathiazole for staphylococci.

In 1938, sulfapyridine was introduced and worked miraculously well against the pneumococcus, on which sulfanilamide had little effect. The mortality of pneumonia was greatly reduced soon after the introduction of this drug. But before long it was noticed that there were many severe and toxic manifestations following its use, especially, nausea, vomiting, hematuria, the formation of calculi, and even uremia.

Then in 1939, another great drug, sulfathiazole, was placed on the market. It was not nearly as toxic as sulfapyridine, and the in-

ternists soon discovered that it was equally as efficacious in the treatment of pneumonia as sulfapyridine. Another great advantage of this drug is that it works well on staphylococci. However, it is not nearly as effectual in the treatment of meningitis as the other sulfonamide drugs because it is impossible to obtain a sufficient concentration of it in the spinal fluid.

In 1940, sulfadiazine was produced and seemed to act well on most of the organisms—streptococcus, pneumococcus, gonococcus, staphylococcus—and to be the least toxic of all the sulfonamide drugs used prior to that time. After a wide clinical trial, it has been proven that sulfadiazine produces just as good or even better results than the earlier drugs. One great advantage it has over sulfathiazole is that it enters the meninges readily and is preferred in all meningeal infections. This is the drug of choice at the present time, and from the clinical data at our disposal today, we may safely say that sulfadiazine is just as effective in the treatment of infections caused by the above-mentioned organisms as the drugs which were first heralded as being specific for them. In addition, it has the distinct advantage of being less toxic.

After two years' work, both experimental and clinical, upon its toxicity, absorption, and excretion, the majority of workers have come to the conclusion that sulfadiazine has the following properties:

1. It is readily but slowly absorbed from the gastro-intestinal tract.

2. It has fewer toxic manifestations.

3. It can be carried in the blood stream in a much higher concentration.

4. It diffuses readily into the fluids of the body attaining a concentration in the cerebrospinal fluid of about 50 per cent of that present in the blood stream.

5. Its tendency to acetylation apparently does not increase during the course of treatment and its acetylated derivative is more soluble even than the free form of the drug and quite readily excreted by the kidney.

6. It is slowly excreted and disappears from the blood slowly.

In passing, I should like to say just a few words about the following drugs also members of the sulfonamide group, though they are not of great importance to us as otolaryngologists.

1. Sulfaguanidine and succinyl sulfathiazole are two of the sulfonamide drugs used in intestinal conditions. They are given in large doses for a week or ten days before the gastro-intestinal tract is opened for resections or anastomosis. The incidence of postoperative peritonitis has been reduced tremendously since these drugs have been used, as they make the gastro-intestinal tract practically sterile.

2. Dodecanol sulfanilamide and promin are used in tuberculosis.

3. Sulfapyrazine recently came from the Middle West and acts very much like sulfadiazine. It has low toxicity and a wide scope of action.

There is no question that these drugs have been a great boon to the otolaryngologists in the treatment of severe infections of the nose and throat. It would take volumes even to mention the numerous and varied instances of infection in which this group of drugs is now producing such spectacular and obvious results. One particular group of five cases impresses me very vividly, in which there was infection of the upper lip and alae of the nose, with a resulting cavernous sinus thrombosis and death. I saw this some years ago, before the introduction of this new chemical.

Consider this history as against the low mortality now observed in infections of the upper lip and nose in which the drug is properly and rigorously administered. The outlook at the present time is more favorable, and the mortality much less than it formerly has been. Naturally the drug in this type of infection should be administered before cavernous sinus thrombosis has developed, and therefore our high rate of recovery is due not to the curing of the cavernous sinus infection itself, but rather to the curing of the infection of the lip or the nose.

At this point I wish to mention the report by Schall[1] of Boston in which he gave an account of the recovery in 3 consecutive cases, from full-blown cavernous sinus thrombosis in which he used the sulfonamide drugs in conjunction with heparin. This is a decided advance in therapy when we remember that Grove[2] came to the conclusion that septic cavernous sinus thrombosis was 100 per cent fatal.

Most acute infections of the throat respond amazingly well to the proper use of this drug, particularly if they are seen early before an actual abscess has occurred. And I think that all of us are cognizant of its value in such conditions as acute fulminating tonsillitis and similar conditions which form such a large part of our work. The general practitioner has been quicker to recognize the value of these drugs and

probably prescribes them more frequently than the specialist, although it is possible that the cases treated by him are more carelessly chosen.

We must always keep in mind the value of these drugs in other infections involving the nose and throat, such as deep infections of the neck, erysipelas, Ludwig's angina, suppurative sinusitis, laryngotracheal bronchitis, edema of the larynx and meningitis.

We must be less enthusiastic in their use alone where there is actual abscess formation, such as retropharyngeal abscess or orbital abscess. However, in spite of the above-mentioned facts, it must always be borne in mind that these drugs do not take the place of surgery and while there are scattered reports of apparently surgical cases getting well without surgical intervention, we know from actual experience that the greatest efficiency of these drugs is obtained when they are used in conjunction with proper surgical procedure.

When these drugs are indicated in serious infections they should be given early and in large doses to bring the blood concentration up to the desired level in as short a time as possible. In cases where sulfanilamide, sulfapyridine, or sulfathiazole is given, the concentration should be between 8 and 12 mg. per 100 cc., but in the case of sulfadiazine, it should be higher—between 15 and 20 mg. per 100 cc. In two of our pneumococcus type III meningitis cases, when the patients were very sick, we kept their concentration up to 40 and 45 mg. per 100 cc. respectively, and they showed no ill effects from the drug, and both patients recovered. Owing to the slow absorption of sulfadiazine (it takes several hours before there is much of it in the blood stream) it is well to give sodium sulfadiazine intravenously, which shows up in the blood in ten or fifteen minutes, at the same time starting sulfadiazine by mouth. The first dose should be 60 or 80 grains, followed by 15 or 20 grains every four hours. In very bad cases 60 grains every six hours should be given intravenously and the same amount by mouth for the first few doses. As the infection subsides the dosage is cut down.

For the past year, it has been our custom in laryngectomy cases to give 15 grains of sulfadiazine every four hours for a week before the operation. This seems to cut down the suppuration and infection in the neck considerably. We have also discontinued the through-and-through drainage in the neck in these cases. Instead, the wound is left open and sulfanilamide powder is dusted into it. This acts better than sulfathiazole as the latter cakes too much. One piece of large plain packing is placed in the wound. This is changed daily, and sulfanilamide is dusted in.

Since using the drug in these cases, the wounds clear up much more quickly than formerly, and the convalescence is shortened.

Every physician prescribing the sulfonamide drugs should be familiar with the toxic reactions that may follow their use. The following conditions may occur: hematuria, renal colic, calculi or complete suppression of urine, anemia, granulocytopenia, hepatitis and jaundice, dehydration and acidosis, drug rashes, conjunctivitis, psychosis and neuritis.

The most frequent toxic reaction following sulfadiazine is hematuria, although there may be more serious kidney complications, such as anuria, calculi or even uremia. The incidence of kidney damage during sulfonamide therapy can be greatly reduced by forcing fluids and alkalizing the urine, because the sulfonamide crystals are more soluble in alkaline urine. Leukopenia and granulocytosis occasionally occur following the use of sulfadiazine. When these occur, stop the drug.

In all severe infections, use the drugs boldly without too much fear of the toxic complications, as only about 10 per cent of the cases receiving the sulfonamide drugs have any complications. Give adequate fluids. Formerly, fluids were restricted, but we now know that a considerable amount may be given, providing the concentration is up to the proper level.

CONCLUSIONS

1. Do not use the drugs in any condition in which dangers of toxicity are greater than the dangers of the disease.

2. Give large doses boldly and early in the disease.

3. If no improvement is noted in 72 hours, stop the drug.

4. Have blood concentration, blood count and urinalysis done every 48 hours.

5. These drugs are a valuable adjunct to surgery but are not to take the place of surgery.

121 EAST 60TH STREET.

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XXXII

SOME FUNCTIONS OF THE NON-ACOUSTIC LABYRINTH

(An Experimental Study)

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Every otologist examines patients in which caloric and rotation tests reveal an absence of labyrinthine function. These patients may exhibit no signs of otic deficiency, aside from a loss of hearing. There is no vertigo, nystagmus, nausea or disturbance of equilibrium, such as is usually associated with an acute labyrinthitis or with a sudden destruction of the labyrinth. Apparently compensation has taken place. Few otologists have had the opportunity of observing many uncompensated cases. Now, the situation is changed. First, the fenestration operation has passed beyond the experimental stage and in trained hands has become a recognized form of therapy for the improvement of hearing. Each successful fenestration operation produces, of course, an associated transitory labyrinthine disturbance. Secondly, head injuries are common in modern warfare. Among such injuries many instances of destruction of the labyrinth may be expected. These observations suggest that a knowledge of the physiology of the labyrinth will become of increasing importance to the otologist.

Numerous studies have been carried out upon the non-acoustic labyrinth by clinical and experimental investigators. These were initiated by Purkinje[1], who in 1820 noted vertigo and nystagmus in rotated, insane patients and incorrectly attributed these phenomena to the displacement of the brain. The relation of such phenomena to the function of the vestibular apparatus was indicated by the publication of Flourens' (1824)[2] experiments on pigeons. One of the first

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From the Department of Physiology, College of Physicians and Surgeons, Columbia University.

Read before the Centennial Anniversary Meeting of the State Medical Society of Wisconsin, Madison, September, 1941.

This work was carried out while the author was a Resident in Otolaryngology at the New York Eye and Ear Infirmary.

mammalian experimental studies was that of Bechterew[3], who in 1883 showed that intracranial section of the acoustic nerve in the dog produces disturbances in equilibrium. A few years later Biehl,[4] working upon the sheep and the horse found that sectioning of the vestibular branch of the VIIIth nerve gave rise to disturbances of equilibrium, whereas similar injury to the cochlear ramus was without such effect. It is this work which provides the rational basis for one of the modern treatments of Ménière's syndrome.[5] With the exception of certain ocular sympathetic deficiencies[6] the effects of a labyrinthectomy are identical with those which follow sectioning of the VIIIth cranial nerve.

The common method of investigating the function of the mammalian labyrinth has been to study the functional deficiencies which follow unilateral and bilateral labyrinthectomy. The earlier methods by which labyrinthectomy was performed on cats are cumbersome, require assistants and cause considerable trauma to the surrounding tissues. After performing a number of labyrinthectomies by the bulla approach of de Kleyn[7] and by the occipital route used by Camis[8] and by Wilson and Pike,[9] the author devised an approach by way of the external auditory canal and tympanic membrane. This last method, which has been called the transtympanic, greatly simplifies the procedure.

METHODS

Healthy cats were kept in the laboratory at least one month before experimentation. All operations were performed with strict aseptic precautions upon cats anesthetized with nembutal (36 mg. per kg. of body weight, intraperitoneally). In different series of animals, labyrinthectomy was carried out by three different methods: the bulla, the occipital, and the new transtympanic approach. Thus an evaluation could be made of the results obtained by the various methods.

The transtympanic method[10] of destruction of the labyrinth may be described briefly as follows. An incision was made behind the pinna exposing the temporal muscle and the cartilaginous auditory canal. The canal was incised, exposing the tympanic membrane. A small dental chisel was passed through the tympanic membrane and inserted into the round window, and the promontory was fractured. The contents of the labyrinth were exenterated by reaming the chisel around. The characteristic crunching sound which was heard when the labyrinth was first entered disappeared as evacuation of the laby-

rinthine cavity proceeded. Anatomical continuity was restored by suturing the pinna in place.

RESULTS

The results have been grouped under the following headings:

1. After unilateral labyrinthectomy,
2. After bilateral labyrinthectomy in one stage,
3. After bilateral labyrinthectomy in two stages.

After unilateral labyrinthectomy. After this procedure certain modifications in function have been noted in a series of 50 cats, of which 31 were prepared by the bulla approach, 6 by the occipital method, and 13 by the transtympanic operation.

The head was turned, so that the jaw approached the shoulder on the side of the lesion. There was also a rotation of the head about the naso-occipital axis so that the operated ear was lower. All of the animals showed a transitory turning and a permanent rotation of the head.

It is of interest to note that the position of the head was independent of the eyes, for the application of a head cloth was without influence upon it. The same head posture was shown by a decerebrated cat with one labyrinth destroyed. Magnus and de Kleyn[11] and Prince[12] have demonstrated that bilateral sectioning of the posterior roots of the upper three cervical nerves is without influence upon the head posture produced by unilateral labyrinthectomy. These findings indicate a direct action upon the head posture resulting from the asymmetry produced by unilateral labyrinthine destruction.

The body as well as the head showed changes in posture. First, there was a temporary body concavity on the side of the lesion, which like the head turning soon disappeared. Second, rotation of the trunk to the operated side was apparent. The trunk rotation was markedly increased when the animal was suspended in the air by its pelvis. The resulting spiral rotation of the body is a direct effect of the labyrinth upon the body, for holding the head symmetrically or bilateral sectioning of the posterior roots of the upper three pairs of cervical nerves is without influence upon it.[11] These facts suggest that the removal of inhibitory tactile and proprioceptive stimuli brought about by holding unilaterally labyrinthectomized cats in the air produces an increased rotation of the head and the body to the side of the lesion.

Differences in the muscle tonus of the two sides of the body can be tested by determining the ease with which the body can be displaced. Although other workers have described a decrease in extensor tonus of the limbs upon the side of the lesion, the difference between the two sides was questionable in the animals studied in this investigation.

A more obvious phenomenon of practical significance is the influence of the neck reflexes upon the limbs.[11] This factor is of considerable importance, for head rotation is a permanent effect of unilateral labyrinthectomy. The head rotation produces a decrease in the extensor tonus of the limb muscles upon the side of the lesion (occiput limbs) and an increase in the tonus of the extensor muscles of the sound side (jaw limbs).

Cats were able to stand the day after unilateral labyrinthectomy. However, when the animal attempted to stand, it often fell to the side of the lesion. The cat may support itself by leaning the operated side against the wall. The legs on the side of the lesion were adducted, whereas those on the sound side were abducted. The tendency to turn toward the side of the lesion disappeared within a few days. Unlike rabbits, unilaterally labyrinthectomized cats never roll. The ability to walk improved daily so that by the end of a week the animal walked fairly well. When such cats ran, they commonly showed a lateral deviation of the body to the side of the lesion without change in the direction of the body axis. These observations are in agreement with those of Magnus and de Kleyn.[11]

The interesting reaction of the rebound phenomenon was demonstrated by suddenly releasing the head which had been previously turned so that the intact labyrinth was lower. There was a rebound with threw the animal violently to the side of the lesion. The rebound phenomenon disappeared in three to four days. There was no response when the head was turned so that the operated ear was lower. The resistance to rotation toward the sound side and the strong over-action suggest an unopposed tonic activity of the good labyrinth. This phenomenon is absent in the frog, for Tait and McNally[13] demonstrated that the original asymmetry can be overcorrected so that the animal actually leans toward the sound side. As soon as the head suffers an effective displacement, whether by spontaneous activity of the frog or otherwise, the previous asymmetrical posture of the head reappears. Since Ross'[14] electrical records from the nerve fibers of the utricular macula show that in the absence of head movement the utricular receptors are inactive, Tait[15] has concluded that once the labyrinth with its utricular otoconia has settled to rest it ceases to func-

tion. This conception of the absence of tonic activity of the labyrinth does not appear to apply to the cat.

Fisher and Muller's[16] observation that a unilaterally labyrinthectomized cat freely falling through the air rotates about the long axis of its body toward the side of the lesion was confirmed. Whether such an animal was dropped freely through the air, was suspended in the air by a cord around its pelvis, or was placed in water, its head and body always rotated toward the side of the lesion. This was a permanent phenomenon.

A successful labyrinthectomy in the cat always produced a definite miosis and a partial paralysis of the nictitating membrane on the same side.[6] After one to four weeks the pupils became equal and the nictitating membrane was retracted.

Eye deviation was a very transitory phenomenon and had passed away before the animal completely recovered from the nembutal anesthesia. In a few cats in which ether anesthesia was used the observations of Mangus and de Kleyn[11] and Camis[17] were confirmed. The deviation consisted in a displacement of the homolateral eye downward and of the contralateral eye upward. Both eyes were deviated slightly to the side of the lesion.

Ocular nystagmus was a significant and consistent finding which appeared as soon as unilaterally labyrinthectomized cats recovered from the anesthesia. It was horizontal in type when the head was in the ordinary position. The quick component was toward the sound ear. The amplitude was greatest immediately after recovery from the anesthesia and gradually decreased in the ensuing days. The duration of the nystagmus was found to be two to five days, usually three or four days. This is in agreement with the observation of Magnus and de Kleyn[11] rather than with that of Fisher and Muller,[16] who observed no nystagmus at the end of the second day.

After bilateral labyrinthectomy in one stage. This procedure caused a more marked disturbance in equilibrium than that which followed unilateral labyrinthectomy. On attempting to sit up the bilaterally labyrinthectomized animal fell to either side. Head movement, which was pronounced, was of two types, a horizontal swaying and a vertical motion which frequently caused the cat to strike its chin on the floor. Head movements decreased after the first few days. The vertical type disappeared, but pendular swaying ceased only when the cat was blindfolded. Magnus[18] believed that the pendular head movements were the result of defective visual impressions.

It is possible that in the absence of labyrinthine function optical impulses produced muscular over-action. Periodic violent rocking of the body from side to side occurred so that the animal lay on the floor with the legs spread widely apart. By the end of a week the cat could walk and run on a wide base, but in a zigzag manner. The head turned from side to side with a corresponding change in the deviation of the body's movement. This phenomenon was noted by Magnus[18] more than a year after bilateral labyrinthectomy.

Head rotation and head turning were not present. Spiral rotation did not occur when the cat was suspended by the pelvis. Instead, the head was held in the midline position with the neck flexed. Head righting did not appear if the animal was blindfolded. The rebound phenomenon was not present in bilaterally labyrinthectomized cats. The head could be rotated to either side without meeting resistance, and no rebound occurred when the head was released.

Eating presented certain interesting features in these animals. The violent head movements during the first two or three days after a bilateral labyrinthectomy made eating almost impossible. However, if the animal's head was steadied by the observer's hand the cat ate and drank without difficulty. Even after the vertical head movements had disappeared in the resting animal, they reappeared with considerable intensity when the animal was fed.

On jumping or falling from a height a bilaterally labyrinthectomized cat did not break the fall with its legs but landed heavily on the body or head. In time the animal learned to right itself in the air so that it landed upon its feet like a normal cat. When the animal was blindfolded it lost this ability. Apparently either labyrinthine or optical impulses are necessary for a freely falling cat to right itself[19].

Bilateral miosis and protrusion of the nictitating membranes were present. Ocular nystagmus did not occur when both labyrinths were destroyed at one time.

After bilateral labyrinthectomy in two stages. The time interval between labyrinthectomies caused a variation in the results obtained. The functional deficiency was less severe the longer the time interval between the two operations. Magnus and de Kleyn[11] state that if the second labyrinthectomy is done within four days of the first operation the results are the same as those which follow simultaneous destruction. If the time interval between the labyrinthectomies is sufficient for the establishment of central compensation, the destruction of the second labyrinth produces symptoms which are the mirror image of those which followed the first operation. Thus, when the time in-

terval between labyrinthectomies is eleven days or more an ocular nystagmus occurs as though the first labyrinth had not been removed.

Whether the bilateral labyrinthectomy is done in one or two stages, compensation takes place slowly. After several weeks, however, such an animal is recognizable only by the fact that it walks with the legs widely abducted and with considerable head swaying.

DISCUSSION

A new method of labyrinthectomy has been described. This operation simplifies the procedure and has not the undesirable postoperative complications which follow labyrinthine destruction by the older methods. The functional deficiencies described are in general a confirmation of the work of other investigators.

Disturbances in equilibrium and ocular nystagmus are two of the most significant results of unilateral labyrinthectomy. Following a one-stage bilateral labyrinthine destruction, nystagmus does not appear, but the disturbances of equilibrium are more marked. However, if a certain time interval separates the two operations the results of the second labyrinthectomy are the mirror image of those which follow the first operation. The disappearance of the ocular nystagmus which follows unilateral labyrinthectomy is usually explained as central compensation. According to Spiegel and Demetriades[20] the site of the compensation is subcortical. The nystagmus which appears when the second labyrinth is destroyed some days after the first labyrinthectomy is called Bechterew's compensatory nystagmus. It appears to be related to the integrity of the vestibular nuclei, for the destruction of these structures abolishes the nystagmus.[20]

These observations have led to the conception that each labyrinth is the functional antagonist of its mate. When one labyrinth or the vestibular nuclei of one side are stimulated or destroyed, the normal balance between the two labyrinthine systems is lost and nystagmus and disturbances of equilibrium result.

CONCLUSIONS

1. The transtympanic method of labyrinthectomy in the cat simplifies the operation and produces the same results as the older methods.

2. Unilateral labyrinthine destruction causes changes in the posture of the head and body, the gait and the reaction to free falling. A nystagmus in which the quick component is toward the sound ear

is present for two to five days. The rebound phenomenon suggests an unopposed tonic activity of the sound labyrinth.

3. Bilateral labyrinthectomy produces more profound disturbances in equilibrium than does the destruction of only one labyrinth. Compensation occurs so that in a few weeks the animals show few abnormalities except certain variations in gait and head movements. Blindfolded bilaterally labyrinthectomized cats do not right themselves when freely falling through the air. Although no ocular nystagmus results when the two labyrinths are destroyed simultaneously, nystagmus does occur when a certain time interval separates the two labyrinthectomies. The type and direction of this nystagmus is the same as if the first labyrinth were still intact.

The author is indebted to Dr. Walter S. Root, Associate Professor of Physiology, Columbia University, for assistance and encouragement in carrying out this investigation.

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XXXIII

THE PRACTICAL MANAGEMENT OF HEADACHE

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The subject of headache is a large one. It is the purpose of this paper to examine only one small but important phase, namely the practical management of the case in hand.

The mechanism of headache, so much as is known of it, has had full discussion in the literature and will be touched upon only as it suits our purpose.

There is no generally accepted classification of headache, although various ones to be found in recent publications fairly parallel one another. These, also, will be disregarded for the moment, not in the least to discredit them but simply to avoid confusion with this present loose and probably unscientific classification, intended entirely to expedite practical management.

While the local mechanisms which produce headache are of only two or three types, many remote conditions provoke them by activating these mechanisms. In general it may be said that all headaches must be the result of disturbances transmitted through the sensory cranial nerves. Experimental evidence exists to show that only certain of these are commonly implicated. There is further evidence that pressure, distortion and other physical insult is painful only when applied to certain restricted areas of the brain and its coverings. Without going further into the matter the sensitive structures may be designated as: the dural arteries, the cerebral arteries at the base of the brain, arteries outside the cranium, the great venous sinuses and the basal portions of the dura itself. Structures said to be relatively insensitive to pain are: the cranium (including the diploic and emissary veins), the parenchyma of the brain, most of the dura, most of the pia arachnoid, the ependymal lining of the ventricles and the choroid plexuses[3].

Adapted from lectures before the Post-Graduate Assembly, Southern Section Meeting of the Texas Medical Society, Houston, Tex., Dec. 1, 1942; the Chicago Laryngological and Otological Society and the Chicago Ophthalmological Society, Chicago, Ill., Feb. 8, 1943; and the Iowa State Medical Society, Des Moines, Iowa, April 29, 1943.

The sufferer from headache visiting the doctor is often at a disadvantage, particularly if the doctor is a specialist. The conscientious specialist, to be sure, keeps himself reasonably informed of the more general advances in other branches of medicine. The causes of headache unfortunately seldom fall into these general categories but tax the diagnostic acumen of each specialist in turn. As a result, instead of telling his patient, "There are thirty odd causes for headache; go to someone who can pick the one that applies to you," the ophthalmologist is apt to say, "It isn't your eyes, it's probably your sinuses," and to send him to a rhinologist who says, "There is nothing wrong with your sinuses, better see a dentist." And so on, to the increasing perplexity of the patient.

What to do?

For purely clinical purposes the following approach has proven more useful to me than any other I have thus far attempted.

The first and most important step is to project the given case into one of the following classifications:

Having (A) Definite demonstrable causes,

(B) Semi-demonstrable causes,

(C) Undemonstrable or only remotely suggestive causes.

It will quickly occur to anyone that such a classification will be largely personal and will depend upon the skill and experience of the diagnostician; what is "definitely demonstrable" to one may be only "remotely suggestive" to another. No matter. It is a tool, a filing system to expedite management, and overlapping is not important.

Under the "A" grouping come, first, local conditions which include demonstrable eye, ear, nose, brain and dental lesions; specific nerve affections such as trigeminal nerve pains and nasal ganglion syndromes; injuries; tumors and infections. Under the same grouping come, second, definite remote conditions such as constipation and other digestive disturbances; organic diseases whose characteristic toxic or mechanical aberrations produce referred pains in the head; allergic conditions definitely traceable to known antigens; anemias; histamine poisoning.

In my own experience the percentage of histamine headaches has not been nearly as high as in the series reported by Horton[1], but some typical cases have been encountered, and the condition being demonstrable belongs properly in this class.

Examples of Class "A"—Definitely Demonstrable Causes

Local	Remote
Injuries	Hypertension
Inflammations	Cardiac, renal or other organic disease
Abscesses	Constipation and related digestive disturbances
Tumors	Organic nervous diseases
Eye strains and diseases	Anemias
Ear disease	Abdominal and pelvic tumors
Nasal disease	Definitely associated allergy
Dental disease	Histamine poisoning
Neuritis and neuralgia	Cough (persistent, violent)
Temporomandibular distortion or disease	Metabolic disturbances
	Infectious diseases
	Alcohol, tobacco, drugs

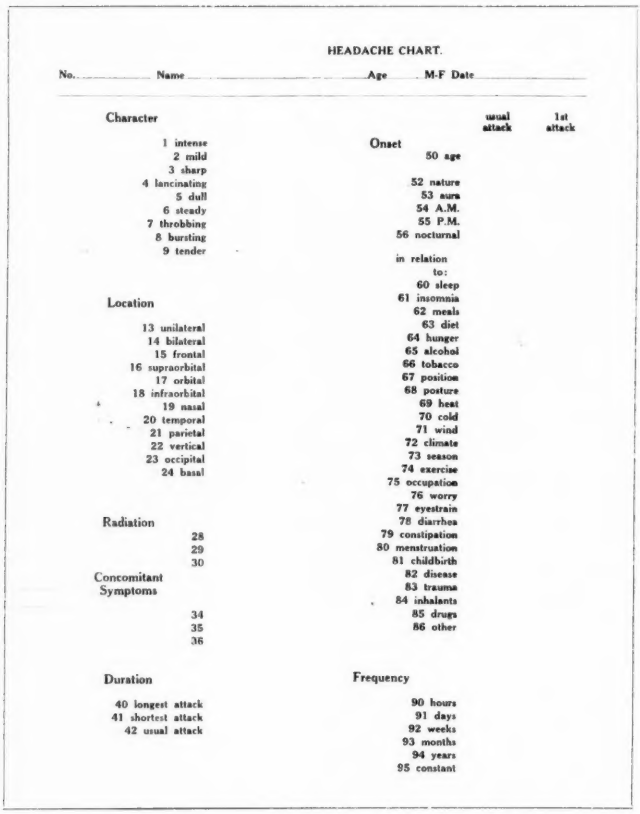
Examples of Class "B"—Semi-demonstrable or Suggestive Causes

Psychoneurotic disturbances
Migraine
Fatigue and asthenia
Occupational influences

Class "C"—"Undemonstrable" Causes. (That is, causes not revealed by examination, by laboratory tests or as part of a recognized syndrome. Idiopathic. If these could be individually designated in a table, they would *ipso facto* come under "A" or "B".)

Under the "B" grouping are migraine, physchoneurotic disturbances, fatigue and kindred relatively definite processes.

Under the "C" grouping come headaches which might once have been described as "idiopathic." They appear to be due to vascular changes occasioned by all manner of influences, and arise probably from acute distentions and more particularly rapid collapses and spasms of blood vessels, alterations in blood pressure, in fact any changes in local vasomotor tone. These merge, often indistinguishably, into the neuralgic and the toxic pains. The three are closely associated with one another and sometimes with more easily demonstrable lesions such as arthritis and arteriosclerosis.



HEADACHE CHART.

No. Name Age M-F Date

Character		usual attack	1st attack
1 intense	Onset		
2 mild	50 age		
3 sharp			
4 lancinating	52 nature		
5 dull	53 aura		
6 steady	54 A.M.		
7 throbbing	55 P.M.		
8 bursting	56 nocturnal		
9 tender	in relation to:		
	60 sleep		
Location	61 insomnia		
13 unilateral	62 meals		
14 bilateral	63 diet		
15 frontal	64 hunger		
16 supraorbital	65 alcohol		
17 orbital	66 tobacco		
18 infraorbital	67 position		
19 nasal	68 posture		
20 temporal	69 heat		
21 parietal	70 cold		
22 vertical	71 wind		
23 occipital	72 climate		
24 basal	73 season		
	74 exercise		
	75 occupation		
	76 worry		
Radiation	77 eyestrain		
28	78 diarrhea		
29	79 constipation		
30	80 menstruation		
	81 childbirth		
Concomitant Symptoms	82 disease		
	83 trauma		
34	84 inhalants		
35	85 drugs		
36	86 other		
Duration	Frequency		
40 longest attack	90 hours		
41 shortest attack	91 days		
42 usual attack	92 weeks		
	93 months		
	94 years		
	95 constant		

Fig. 1.—Headache chart, page 1.

Finding the proper place for the case in hand, in terms of this classification, is not simple—is indeed not always possible. The most direct and to me the most reliable method is a standardized headache history, recorded upon special forms. Such a record, in addition to the general history, serves to segregate the findings pertaining specially to headache and throws them into juxtaposition so that their relationships become more apparent. A copy of the form which I find useful is shown in Figs. 1 to 3.

Supplementing this form, I employ also a chart (Fig. 4) kept by the patient for several weeks, on which he records foods and drugs

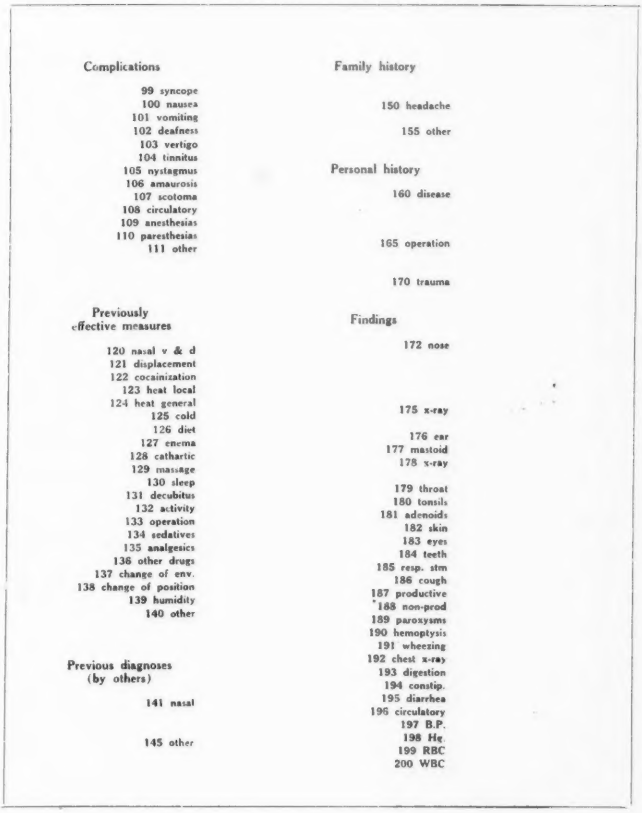


Fig. 2.—Headache chart, page 2.

ingested and some of his more important contacts. Much information is derived from this chart. Aside from its value in cases of allergy, it demonstrates graphically the patient's eating habits, idiosyncracies of diet, intake of alcohol, smoking propensities, and exposure to industrial dusts and fumes.

Being familiar to the examiner these standard charts are much more easily evaluated than the nondescript diaries kept by inexperienced patients. The two records further impress the patient with the importance of detail in arriving at a diagnosis. The second one

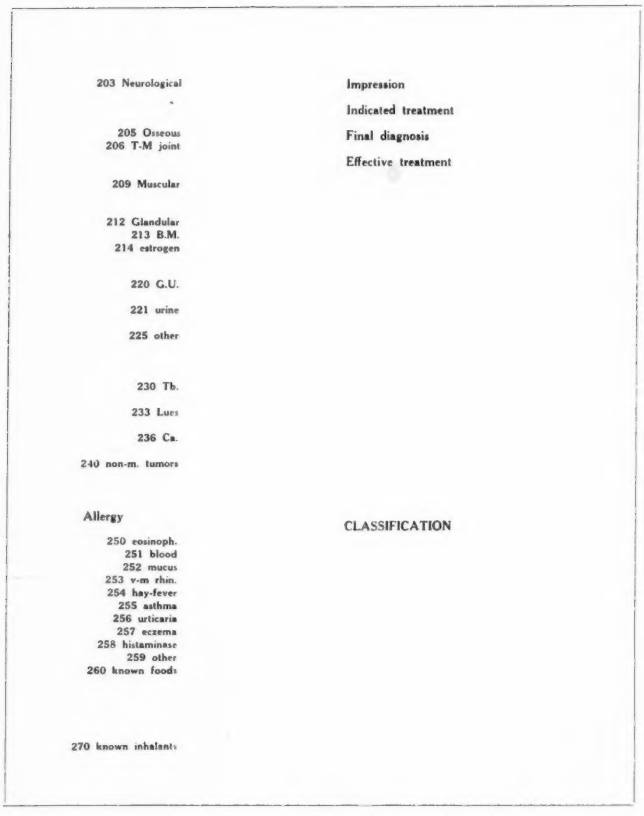


Fig. 3.—Headache chart, page 3.

enlists his daily interest, focuses his attention and brings forth helpful comments of his own.

A complete physical and laboratory examination follows. After such examination class "A" takes care of itself. The remedy is obviously the removal of the cause when that is possible, since Class "A" consists, by definition, of cases having a demonstrable cause. This, as stated before, may be a sinus infection, a hyperphoria, a brain tumor, or any of a great number of disturbances disclosed by the examination. The facility with which it may be eliminated will, therefore, vary.

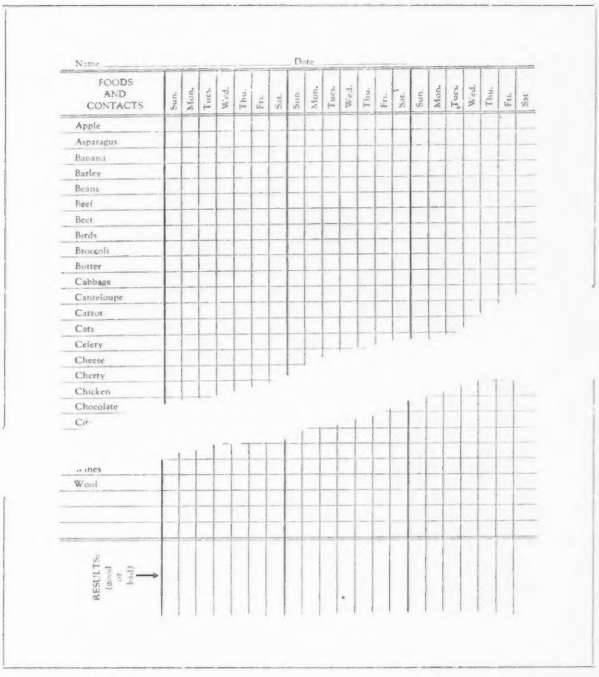


Fig. 4—Food and Contact Diary. The complete chart lists 72 substances.

Patients classified under "B" will be apt to require readjustment in their occupations or some other dominant factor in their daily lives. Their problem is often one for the sociologist. For the poor unfortunate, whose headache turns out to be hereditary, there is not much help. There are those who would have us believe that all persistent, chronic headaches can be laid at the door of our immediate ancestors, and that once we are born it is too late. I am not one of these. For that matter, there are those also who believe that all headaches come from eating onions or lobster or rhubarb. Thus the literature of headache is deeply tinged with the personal experiences of authors, which is natural enough but does not always get us forrader.

Class "C" comprises the most perplexing but at the same time the most satisfactory cases. For this group the classification has been particularly useful. It may be considered the experimental group.

It will be recalled that before launching upon the following more or less experimental measures, the routine laboratory tests, including various allergy tests, determination of the basal metabolic rate and complete blood examinations, have been made. It is assumed that such local procedures as the removal of nasal obstructions, cocainization of the nasal ganglion and the nasofrontal nerve, and opening the ostia by means of displacement with vasoconstrictors, have also been executed when the indications have warranted.

To add to our slender knowledge of the local mechanism of headache, it has been shown that extreme distention of the mucosal linings of the sinuses occurs without pain[2]. Whether this is true when the periosteum becomes involved has not been determined but it is probable that such distentions are painful. We know also that sinus infection does not necessarily mean sinus headache; a great many people have sinusitis and a great many people have headache; the two may exist in one individual and be unrelated. It is important to remember that less than five per cent of headaches are referable to sinuses no matter what their location, and that the great majority are due to local vascular disturbances whatever their remote cause.

It is the opinion of several observers that when pressure changes occur in the vascular structures of the brain, the pain is likely to occur during the relaxation and dilatation which follow pressure, and not during the elevation of pressure.

We begin, therefore, with experimental alteration of the vascular tone. Unless there is hypertension, the patient is given ephedrine orally (together with seconal or some other barbiturate to minimize the unpleasant effects). Many headaches disappear promptly and many are controlled by this means alone. The ephedrine is given in doses of three-eighths grain twice daily and if effective is continued for a week. If, after the drug is withdrawn the headache returns, it may be resumed but a long continued course of ephedrine is not recommended. Some persistent headaches can be controlled with occasional courses of ephedrine. If they are affected at all—even though increased—the suspicion of their vascular origin is confirmed.

If this is unsuccessful, and especially if the patient complains of lassitude and fatigue, thyroid extract takes the place of ephedrine. If in the first general examination the basal metabolic rate was palpably low, then the patient will already have been given thyroid. However, the basal rate often fails us in such cases and experience has shown that even when this is "normal" or slightly increased thyroid may still defeat the headache and cautious experimental administration is

indicated. If this succeeds, then the dosage is subsequently determined by the symptomatology rather than the metabolic rate. These patients are apt to complain of deep boring or bursting headache and there may be sensitivity of the anterior deep temporal artery.

In this connection should be mentioned a recent observation of Williams and Kendall[4] to the effect that the activity of the thyroid is related to the thiamine intake and that thyroid may be better tolerated if thiamine is administered at the same time.

The third experimental measure consists in evacuating the lower bowel, preferably with an enema, at the very onset of an attack, regardless of the patient's statement that his habits are regular. We are all familiar with headache, usually basilar or occipital, resulting from obvious constipation, but similar headaches can be produced by some local retention or possibly by the absorption of toxic products without demonstrable stasis. Abdominal discomfort often announces, or is coincident with, the headache and has been described as of allergic origin.

Minor episodes of starvation—especially habitually recurring ones such as may be caused by too long a period between breakfast and lunch—can produce typically basilar and occipital headaches. It should be pointed out that although these pains may be prevented by the taking of food before they begin, once they occur, the ingestion of food will not diminish them; in fact, they may persist for several hours in spite of it. This is important to remember in arriving at a diagnosis. The remedy lies in a bite to eat between meals, with an eye to the choice of food.

Several other causes of headache remain which must be discussed individually with the patient and which fall into no particular pattern.

In regard to the ingestion of alcoholic drinks it is important to determine not only the amount ingested but the time in relation to eating, sleeping and exercise, and the type of drink, also the time relation between drink and pain.

Smoking habits must be determined. This also includes not merely a question of amount, but any individual idiosyncracy such as a personal hypersensitivity to tobacco or the practice of blowing smoke violently through the nose, or deeply inhaling it. Local irritations from tar deposits are especially common in patients with septal spurs or other constrictions, deviations and obstructions, and headaches from such irritations are not uncommon.

Headache may result from sleeping overlong, especially if ventilation is faulty.

Lack of humidification in heated houses causes a parched membrane and produces a characteristic boring type of pain, usually referred to the forehead and the bridge of the nose.

Faulty posture in typists, pianists and technical workers produces pain in the occiput and shoulders which is sometimes persistent for a time after the condition is corrected.

It is well to remember that a headache—the same headache—may at times result from one cause and at times from another. Until both causes are eliminated complete relief will of course not be attained.

It will be seen from these few examples that while Class "A" and Class "B" are largely bounded by the facilities of the clinic and the laboratory, Class "C" is limited only to the scope of the examiner's ingenuity and powers of observation in the individual case.

It may be added that there is a retribution which attaches to any slight success in the treatment of headaches: grateful patients send their suffering friends, who without exception appear to gravitate unaccountably but inevitably into a final Class "D"—intractable and incurable.

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XXXIV

THE PALATINE TONSIL IN THE SIXTH DECADE*

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Amidst the well-coordinated cyclical sequence of evolution and involution of the whole organism, the lymphatic tissue takes a remarkably independent course. According to Cowdry,[5] the lymphatic system may be regarded as probably occupying a very central position in the phenomena of aging. The tonsils, as part of the lymphatic tissue—but with peculiarities in structure and much exposed to external influences—seem to be even more independent in following their own pattern. As soon as one tries to gain a clear picture of the appearance and the behavior of the tonsils in their own involution, or, on the other hand, of the role of the tonsils in the involution of the organism, many questions arise. The pharyngeal tonsils persist after puberty only as an exception, but this is by no means the case with the palatine tonsils.

The difficulties of distinguishing between normal and pathological conditions arise equally with macroscopical and microscopical observation. There is no gross feature such as size, surface color, surface differentiation, consistency or type of adhesion to the lateral pharyngeal wall, which would clearly divide the normal from the pathological. In the histological picture, many details originate as reactions to morbid agents, so that no normal basis for comparison is present. Except in malignancy, very large abscesses and some other rare conditions, no biopsy findings may be taken as justification for operative measures. This circumstance is responsible to a great extent for a peculiarity in the operative indications. While, even with rigid selection, tonsillectomy on a child remains one of the surgical procedures which is executed with the greatest frequency, in advanced age there is a resistance on the part of the patient and a reluctance on the part of the physician toward the same operation. This is so in spite of two facts: (1) that the indication is in many cases as urgent in later as in younger years and (2) that the intervention itself is simpler with the advancing age of the patient. These two points were emphasized

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in a previous publication[18] and the main scope of the present investigation is to try to furnish additional data concerning this question.

The problem of tonsillar involution has been handled by many authors, usually as a by-product of a general investigation of the tonsils. The result is an enormous amount of information with two outstanding characteristics: contradictory findings and data taken for granted without proper reexamination. The incoherence of the mass of assertions is increased further by the above mentioned discrepancy between the life cycle of the tonsil and the organism as a whole. Restricting the tonsillar findings to a certain age group seems to be a way to eliminate the life cycle of the whole organism as an experimental variable. Only by detailed work of this kind may one arrive at a more exact general picture.

Whether preference should be given to operative material or organs taken from cadavers is a question that can be answered in different ways.

Material can be taken from cadavers with less injury and the block of tissues can include the adjacent structures of the tonsils. Undoubtedly, it is a great advantage to examine the organ in its environment.

But the tonsil in the cadaver by no means represents the normal condition because lethal diseases often produce nutritional changes in the last days *ante mortem* causing considerable depletion in the size of the lymphatic tissue. Beside this quantitative objection the qualitative is of even greater importance. The disintegration of the lymphatic tissue in the cadaver is so rapid that for want of operative material some authors were compelled to use the tonsils of animals to obtain organs fresh enough to render all the staining reactions in desired perfection.[8] The secondary nodules decay very quickly.

In spite of the disadvantage of inevitable injury caused by the grasping instrument, operative material presents the possibility of immediate fixation, a circumstance of paramount importance.

The material discussed here was furnished by 50 pairs of tonsils removed by operation from individuals between the ages of 51 and 63 years, only three of them being over 60. Senescence, according to Lipscomb,[21] begins generally about the age of 55, while others take the age of 65 as the limit. This decade, well at the threshold of advanced age, seemed able to furnish the desired information. Operative indications were the well-known conditions and will not be discussed here. Macroscopically, the highest grade of involution was presented

by the aspect of a mere granulation within the tonsillar fossa, the very last stages showing under the microscope only scattered accumulations of lymphocytes in a scarlike tissue. Even these small remnants caused symptoms necessitating their removal in strictly evaluated cases. As discussed in the before mentioned publication,[18] tonsillectomies in the more advanced age group are characterized, contrary to the opinion of several authors, by remarkably slight hemorrhage and a rapid and uneventful recovery with a minimum of discomfort for the patient. The impression given by the earlier, smaller group, was entirely confirmed by this larger one of 50, including 21 men and 29 women.

Ten sections were cut from each tonsil and, after formalin-fixation and paraffin-embedding, were stained as follows: hematoxylin-eosin; Masson for connective tissue; Verhoeff-Van Gieson for elastic elements; Gömöri's impregnation for the fibrillary work in the reticulum; Maximow's azur II for cytological details, bacteria and fungi. The sections were made in the greatest diameter of the tonsil, in a generally frontal plane.

In the following data the total does not always reach a hundred per cent as some sections did not show all the details. Besides this, percentage figures would not mean much where only complete serial sections would give the answer.

Absolute and relative size. The outstanding macroscopic feature of involution being reduction in size, these data may be duly put at the beginning. Considerable reduction in size is caused by fixation in formalin and a correction should be made in this respect.

The absolute size of the tonsil was noted and then compared with that of the contralateral organ.

TABLE 1.—ABSOLUTE SIZE

Large to very large	24
Medium	23
Small to very small	51

The relative size is discussed in connection with the stage of development. The tonsils were divided into three main groups. The first was the fully-developed tonsil, as it is seen at the climax of its evolution. The second showed distinct signs of beginning involution, that is, eversion of the crypts in lobules with and without connection with each other. The third contained findings of reduction of the organ to the size of the tissue surrounding a single crypt, or to a simple

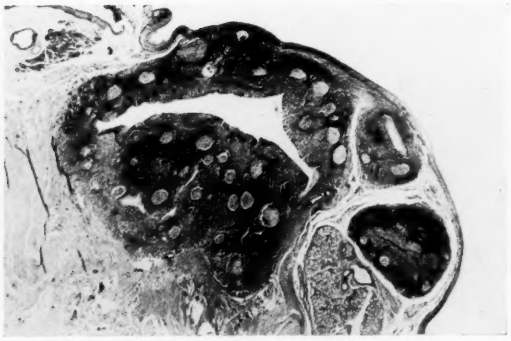

Fig. 1.—Hematoxylin-Eosin Stain, x30. Tonsil reduced to a single open crypt and adjacent structures.

lining of the pharyngeal wall with tonsillar tissue, or even to a mere diffuse infiltration of a part of the tonsillar fossa with lymphatic tissue which was not organized into tonsillar structure.

Twenty-two pairs of tonsils were found to be of equal size and structure, whereas in the remaining tonsils the differences are set forth in the following table.

TABLE 2.—SIZE AND STRUCTURE, RELATIVELY ON BOTH SIDES

	ONE SIDE	BOTH SIDES	CASES (TOTAL)
Full development	11	12	23
Marked involution	8	8	16
Maximal disorganization	7	15	22

Figure 1 shows a tonsil, reduced to a single crypt, which of itself does not present any difference from a crypt in a fully developed tonsil. The epithelium of the free surface is intact; the capsule, the septa, the crypt lumen, the crypt epithelium and the secondary nodules show the normal disposition. This is a purely quantitative reduction inasmuch as the whole tonsil is represented by this single crypt and its surroundings.

The next stages are a closed crypt, and one with signs of beginning eversion. This process begins with a dilatation of the mouth of the crypt at the external surface and will go on, until a funnel-shaped

cavity is presented and finally an open shallow fossa. Around the mouth of this newly formed, flat depression, the everted epithelium of the former cryptal wall is already forming the surface epithelium. With progressive flattening of the everted crypt the last stage is represented by a flat layer of lymphatic tissue (Fig. 2) beneath the former cryptal epithelium, lining an area of the pharyngeal wall. At this period, when the primary nodules, that is, the lobular subdivision of the tonsil, are already gone, the structure of the secondary nodules remains generally well preserved. The very last condition is seen where only islands of diffuse lymphocyte aggregations can be distinguished amidst the connective tissue of the pharyngeal wall under the surface epithelium.

Simple disappearance of the crypts with their respective surroundings, may be termed type 1, as distinguished from type 2, which is characterized by eversion of the crypt in such a manner that its epithelium becomes a lining of the free pharyngeal surface. This latter may be the only final form of every tonsil before total regression. It is possible that type 1 always has to go through a stage like type 2 on its way toward the end.

Connective tissue. In discussing the connective tissue, the best way is to start with the capsule, as the connective partitions originate from this main source.

TABLE 3.—CAPSULE

	NUMBER OF TONSILS
Continuous, simple	39
Continuous, very thick	12
Interrupted by islands of glands or muscles	6
Partial	8
Disorganized	13
Not present in the sections	10
Hilus formation only	6

TABLE 4.—RELATIVE DEVELOPMENT OF THE CAPSULE ON BOTH SIDES

Equal	14
Different, in grade	17
Different, totally	14

The following table gives information regarding the character of penetration of the tonsils by connective tissue.

TABLE 5.—PENETRATION OF THE TONSIL BY CONNECTIVE TISSUE

Full development	
Parenchymatous tonsil	12
Fibrous tonsil	19
Involution	
Penetration marked	
at center	1
at one pole	10
at both poles	1
General thickening of septa between crypts	13
Conspicuous central septum: Intratonsillar plica completely dividing the tonsil	9
Penetration diffuse over the whole tonsil	8
Complete walling off of islands of lymphatic tissue	8
Connective tissue only as support behind flat lining of lymphatic tissue at the free surface	7
Complete disorganization and blending of lymphatic and connective tissue	8

TABLE 6.—PENETRATION BY CONNECTIVE TISSUE IN TONSILS ON BOTH SIDES

Equal	23
Different, in grade	13
Different, totally	10

The central septum (the intratonsillar plica) divides the organ completely into two parts. Areas of lymphatic tissue become frequently isolated as independent islands but more often the diminishing lymphatic tissue is found as a single mass before the ultimate disintegration.

In connection with the capsule several other findings will be mentioned.

Hemorrhages. These were almost completely absent in 40 tonsils, a minimal or medium amount of blood was found in 47, a considerable amount in 6. As these findings depend entirely on eventualities at the operation, no comment is extended to them.

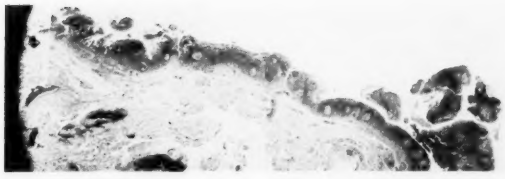

Fig. 2.—Hematoxylin-Eosin Stain, x30. Completely everted crypt-epithelium, lining the free surface.

The same is the case with salivary glands which may or may not be included in the surgically removed tissue. They were present in 62 tonsils, located 7 times only behind the center, and were absent in 31 cases.

Muscle tissue. Muscle tissue was found in different amounts, but this, too, depends usually on the operation itself. Once this tissue was found within the lymphatic tissue, enclosed between two crypts. Once lymphatic tissue was found in an isolated, island-like location behind the retrotonsillar muscle wall.

Elastic fibers. Of importance is the participation of elastic fibers in the formation of the capsule, as it is thought to increase with age. The Verhoeff-Van Gieson stain gave the following results:

TABLE 7.—DISTRIBUTION OF ELASTIC ELEMENTS

(NUMBER OF TONSILS)

	NONE OR MINIMAL	FAIR	ABUNDANT	VERY ABUNDANT
Capsule	12	54	26	3
Septa	44	41	8	—
Submucosa	74	10	3	—

TABLE 8.—DISTRIBUTION OF ELASTIC ELEMENTS ON BOTH SIDES IN 44 PAIRS OF TONSILS

Equal	9
Different, in grade	23
Different, totally	12

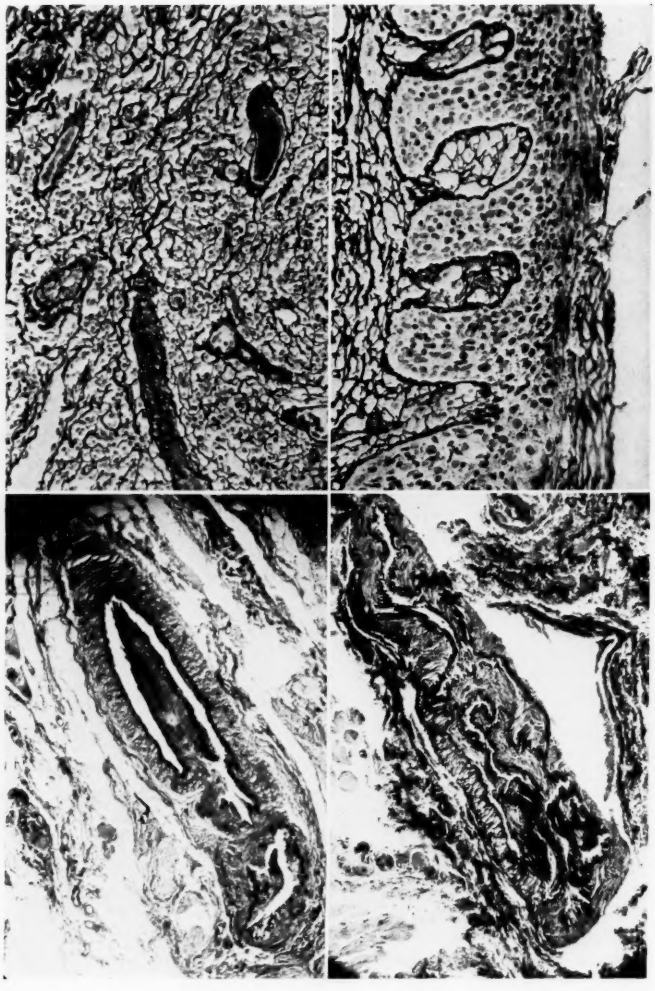

Fig. 3.—Gömöri's impregnation, x550. Fibrils of the basal reticulum, arising from vessel-walls.

Fig. 4.—Gömöri's impregnation, x800. Network of basal reticulum projecting into the epithelium enveloping as support the transmigrating cell groups.

Fig. 5.—Verhoeff-Van Gieson Stain, x600. Capsular artery with ridge-like structure, facilitating rapid caliber-variations.

Fig. 6.—Verhoeff-Van Gieson Stain, x600. Arterial conglomerate in the capsule. Cushion, at center, and several ridge-like formations, narrow down the lumen.

An increase in elastic fibers would mean, beside thickening, penetration as far as the submucous tissue beneath the epithelium of the free surface, but this happens very infrequently. Generally the elastic fibers will penetrate from the capsule, where they are almost always present, into the septum, but not far. As soon as the septum is divided into the finer branches, the elastic fibers do not follow. In most cases they do not penetrate further than the base of the main trunk of the septum. A remarkable aspect of their distribution is the formation of elastic fiber capsules around bone and cartilage pieces, blending with their periosteum or perichondrium.

Hyalinization. True hyalinization is not easily distinguished from scar formation in connective tissue. With the Masson and Van Gieson series at our disposal the mass of penetrating connective tissue could be often interpreted in both ways. It can be safely said that the degree of hyalinization was not sufficiently conspicuous to be characteristic of the involuting tonsil.

The vessels of the capsule. These are of particular interest. Their respective natures and the stage of their development may contribute much to the course of the involutional process. In the arteries, the cushions described by Brunner[1] were found in many instances; when cut longitudinally, they resemble wedgelike formations or ridges, as shown in Fig. 5. Here the long drawn-out ridge is not unlike an ampullar crista. Fig. 6 shows how in another longitudinal section of an artery manifold ridges and cushions cut in several directions help to close the vessel tightly. The elastic membrane is easy to follow in Fig. 7, where two sets of fibers cross each other with muscle fibers between them. Another mechanism for closing the artery is represented by Fig. 8. A sleeve-like portion of the severed vessel is rolled back forming a solid plug with its elastic membrane. The capsules of 14 tonsils contained these cushion arteries; in one instance a similar structure was found in the lymphatic tissue. This number would have increased without doubt in serial sections. Occlusive plugs were found in many small, but thick-walled arteries.

The veins could be classified according to both types described by Brunner, that is, with and without muscle in the wall. With a very low number of thrombosed veins, only once was a band found traversing a vein. This finding represents, according to Brunner, the remnant of a resorbed thrombus.

Arteries and veins built in many instances rich plexuses behind the tonsil, a circumstance the importance of which must be stressed

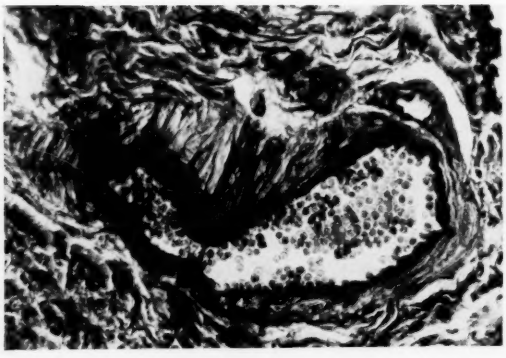

Fig. 7.—Verhoeff-Van Gieson Stain, x600. Crosswise disposition of fibers in elastic membrane beneath the intima, in a ridge of the arterial wall.

because of the variations in size according to the momentary blood supply.

Perivascular round cell infiltrations. These were an infrequent finding. They occurred sometimes in several retrotonsillar areas at a time.

Lymphatic tissue. The lymphatic tissue has to be discussed as to its general disposition and the details of its finer structure. The epithelium, both at the external and at the cryptal surface, is so intimately connected with the lymphatic tissue that they must be reviewed together.

Reporting the findings in the less involuted tonsils would be only an enumeration of well-known characteristics. To avoid this, the following discussion is based exclusively on the study of the 25 tonsils most advanced in involutional disintegration.

The lymphatic tissue appears in three forms; (1) as diffuse infiltration of the connective tissue with scattered, independent lymphocytes, in no connection with each other (such a region may show intimate blending of lymphocytes with collagen, even with elastic fibers); (2) as the homogeneous basal lymphatic tissue around the crypts; (3) as secondary nodules, representing characteristic grouping of the elements of the diffuse lymphatic tissue. In the most

disorganized tonsils all three forms were found together. Beside large areas with simple lymphocytic infiltration similar to the embryonic type, there were islands of coherent lymphatic tissue with secondary nodules. The dark zones at the boundary of the latter were directed invariably against the open space, the cryptal lumina or, after eversion of the latter, against the pharyngeal cavity. Examples of every one of the three types were found in the specimens without the presence of the other two. Solid blocks of lymphatic tissue without secondary nodules sometimes composed as much as half of the whole tonsil. On the other hand, such blocks have been in other cases almost entirely differentiated into secondary nodules, without the presence of crypts.

An attempt was made to measure the size of the secondary nodules with the aid of an ocular micrometer, as this figure is often discussed in connection with the age and activity of the tonsil. As the nodules are cut in every possible diameter, only the largest figures have any value at all, and even these must be multiplied about twice because of the shrinkage. For example, measuring the rosary-like row of 17 follicles of the surface-lining, representing the only remnant of a tonsil as shown in Fig. 2, resulted in an average of 561 microns. This is well within the normal limit.

The activity of the secondary nodules can be studied only in strict connection with the epithelium. The reticulation of the latter is begun by the formation of a nodule beneath the basal membrane. Then this membrane is interrupted, and with infiltration of the epithelium with lymphatic cells, a bulging is produced with final emigration of lymphocytes and granular leukocytes into the cryptal lumina or directly up to the free pharyngeal surface. The reticulation is much more intensive in the cryptal epithelium, whereas the external epithelium may remain uninterrupted for long stretches. Both kinds of epithelium are set off fairly sharply where the surface epithelium dips into the depth of a crypt. With the most characteristic process of the involution (the eversion of the crypt) the cryptal lining becomes the external wall. But even at this stage the two types show a clear boundary which has moved from the cryptal orifice to some point of the external surface. Moreover, the cryptal epithelium, even at this stage, undergoes the same intensive reticulation as before. The original surface epithelium conserves the uppermost layers of condensated, clear, flat epithelium cells with but a few points of interruption by emigrating lymphocytes and leukocytes. There is hyalinization, whereas the former cryptal epithelium maintains its tendency to the contrary process, that is, loosening, and

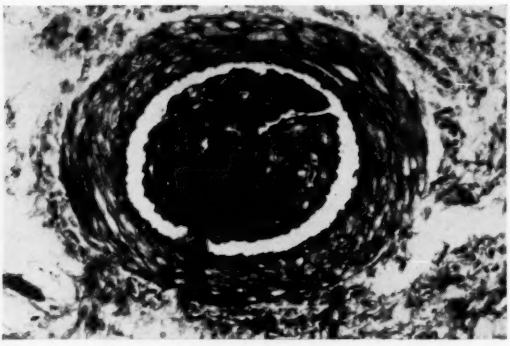

Fig. 8.—Verhoeff-Van Gieson Stain, x600. Plug in capsular artery, representing terminal portion of the severed vessel, rolled back like a sleeve.

swells with desquamation. After eversion, the boundary between the two types of epithelia lies farther away at the free surface instead of at the mouth of the crypt.

Detailed study of the constituents of the basal lymphatic tissue itself did not reveal any difference between a fully-developed tonsil and the last vestiges of the disintegrating organ. Such a remnant, stained with Maximow's azur II, shows groups of penetrating lymphocytes and leukocytes amidst the epithelium. Fig. 4 was obtained by Gömöri's impregnation: the loops of the reticulum fibers are forming a cage around the penetrating cells and demonstrate the undiminished activity of the latter.

Plasma cells. Until the last stage these cells show a definite tendency to appear in groups around the penetrating lymphatic cells, just beneath the basal membrane of the epithelium.

Cysts. Cysts were found in nine tonsils, mostly in the lymphatic tissue. Three were filled with fibrin, four with cholesterin, two were of type Liveriero. They were found more often in disorganized organs than in fully developed ones, in a ration of 7:2. One of them showed a thick wall of lymphocytes in spite of its location far back in the capsule. Lymphocytes penetrated the squamous epithelium and were seen within the mass of cholesterin.

Conspicuous masses of cryptal inclusions consisting of bacteria, fungi, desquamated epithelium and outwandered lymphocytes and leukocytes were present in 19 tonsils, 7 times in well developed organs, 12 times amidst disorganization; once an actinomyces colony filled in the bottom of a crypt.

Bone. Bone was seen in the capsule nine times; in the lymphatic tissue once. Cartilage was seen in the capsule five times; once in the lymphatic tissue. Six of these tonsils were well-developed, ten in regression. A row of pieces of bone and cartilage was found forming part of a bony capsule behind the tonsil. Pieces of this chain, with bone and cartilage closely connected, represented intercartilaginous ossification. A piece of hyaline cartilage showed the peculiarity of being entirely surrounded by the salivary glands of the capsule; finally, Fig. 9 shows a peculiar osteoid formation surrounding the bottom of a crypt like outpoured cement.

COMMENT

The objection has to be met that this operative material is not representative of the true general stage of the organ in this age group. These tonsils must have been uncommonly active if they necessitated an operation to liberate the organism from their influence. But, the presence of every stage of involution among the specimens shows that it is not the more juvenile tonsil which is the more active. How these conditions apply to adjacent and distant parts of the organism was discussed in the previous paper.[18]

After securing general information by examination of the whole series, the 25 tonsils in the most advanced stage of disintegration were used for a closer check-up. This method seems to lead directly to the core of the problem. As is shown in Table 2, the number of cases with marked disintegration on both sides was 23. Because of these 23, together with 15 other cases where this condition was found at least on one side, it can be said that two-thirds of the tonsils were well on the way to regression. Roughly the same ratio is expressed in Table 1. Taking large size as an indication of good development and diminishing size as a sign of disintegration, a ratio of two-thirds appears again for tonsils in involution. This is a surprisingly low number. Even when many authors insist that involution cannot be connected with any definite age, it is generally understood that the process begins early, at the age of 30 at the latest. The ratio of one-third for "juvenile" tonsils is certainly higher than would have been expected for this age group.

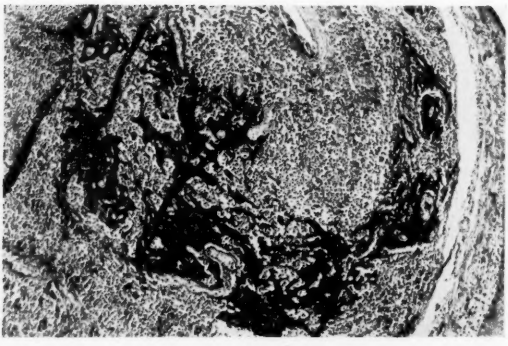

Fig. 9.—Verhoeff-Van Gieson Stain, x200. Osteoid, like poured cement, in lymphatic tissue around the bottom of a crypt.

Wright[20] described an auxiliary process accelerating the atrophy of the tonsils and termed it autoclasis: papillary excrescences appear on the walls of dilated lacunae, as seen by Wingrave, and at the free surface, where they are sometimes called supernumerary tonsils. These structures are the result of degeneration and the pedicle finally breaks off. The patient "is gradually swallowing his own tonsils—piecemeal."

The general picture of the tonsil in involution has been described by many authors. As it is impossible to quote them individually, a summarizing presentation is given based mainly on the data of Barnes,[1] Berggren and Hellman,[2] Cowdry,[5] Dietrich,[6] Giuffrida,[11] Hellman,[14] Liveriero,[22] and others.

The tonsils may show involutionary signs beginning at almost any age, but from 30 years of age on the reduction in size is conspicuous. The organ is reduced to a mass of fibrous tissue as a result of repeated inflammation and irritation. The involution is the same in both tonsils, frequently lobular in form, with atrophy in one or the other lobule. The embryonal disposition may come back, with reappearance of an intratonsillar plica. The final phase of the life history of the cryptal system is reminiscent of late prenatal and early postnatal life; the portion closest to the capsule is the first to degenerate; the antler-like arrangement is lost. The infantile form is represented in the extreme stage by crypts with epithelium everted to the surface. Regarding the mucosa, only with difficulty can cicatrization be differentiated from involution. Goblet cells are more abundant. The lymphocytes are less numerous within the epithelium; formerly isolated epithelium fragments fall together with more or less complete regeneration and a tendency to keratosis. The borderline between epithelium and connective tissue becomes again more intact, as

in the newborn. The cryptal lumina show papillary excrescences; foreign bodies are seen but there is little or no reaction around them. At the orifice of the crypt there is a plug which is composed mainly of accumulated keratoid masses and outwandering lymphocytes; cellular debris is scarce in advanced atrophy. In the parenchyma of the basal lymphatic tissue the vessels are generally increased as is the connective tissue along with rarefaction of the cellular elements. Arterioles show hyalinization and decrease in lumen. The lymphatic tissue may be reduced to a mere strip around the cryptal lumen; the remainder is a solid mass of fibrous tissue which is replaced more and more by inert connective tissue rich in cells. The secondary nodules decrease in number and in size. The number of their cells become smaller, but the nodules are very resistant and disappear only with almost complete regression of the lymphatic tissue. Even with a considerable amount of lymphatic tissue present, the germinative centers are relatively small, or absent. In the activity-center the number of dividing cells is decreased; lymphocytes penetrate toward the clearer center, later causing a profuse infiltration with lymphocytes in this area. The characteristics of a lymphoblastic center disappear; there are fewer lymphocytes, but more reticulo-endothelial elements. Vessels become more numerous and more prominent. The septa of connective tissue form a diffuse pattern without secondary nodules; the lymphatic tissue becomes inconspicuous amidst the fibrous tissue; the latter is relatively increased in amount due to recession of lymphocytes from its fibrous mesh. The increase, at the expense of lymphatic tissue, appears in several main forms: in the shape of irregularly distributed sclerotic processes along the septa, fanlike in a more homogeneous and symmetrical manner from the base to the mucosa of the free surface, and finally shrunken thereby completing the picture of the atrophic tonsil. The capsule becomes indistinct or even unrecognizable.

As against this conception, condensed from the most competent sources, the present investigations resulted only partially in an affirmation. Some of the most important features will have to undergo substantial modification.

It is true that advancing connective tissue pushes apart the formerly coherent lymphatic tissue into isolated islands which later lose their arrangement into crypts. These islands may be seen amidst blocks of hyalinized connective tissue. The reappearance of the intratonsillar plica observed in the embryonic period can be seen in several instances. But these conditions do not represent the general trend followed by the regressing tonsil.

Reduction in size is much more marked in the horizontal diameter. The last remnant is frequently a ridge, long and narrow, running parallel to the plicae. Another type is one that might be called the "drop" form; the ridge enlarges downward, hanging even below the lower end of the tonsillar fossa. It is difficult to say which part of the tonsillar fossa is more frequently emptied first, but it seems that the most reduced remnants are usually found in the lower third. The same degree of decomposition on both sides is found about a third more frequently than cases with divergent findings in each

organ. One or the other lobule may be much further ahead on the way to regression even as at the height of their development the lobules show conspicuous differences. However, no importance can be attached to this circumstance.

The final phase in the life history of the cryptal system is outlined by two types: with and without eversion. In the first type, the last thing to be seen is a solitary crypt which finally melts into the connective tissue; in the second type the cryptal wall through eversion becomes part of the lining of the free surface. In type one the process goes on in the depth, well covered by the intact surface epithelium; in type two, at the surface itself. As the term "tonsil" is given only to lymphatic formations with secondary nodules, the ultimate diffuse form of infiltration with lymphocytes is already beyond this phase. In fact, such aggregations are produced at many points of the pharyngeal mucosa.

There is no rule concerning the order of disappearance of the last elements. The solitary crypt may or may not have a lining of secondary nodules. The last isolated block of lymphatic tissue may be a solid mass of lymph cells and may or may not connect with a crypt. An isolated block may show complete division into a light center and a dark zone, even without a connection with any surface, cryptal or external. It is possible that every crypt ends finally with eversion onto the free surface. None of the specimens showed a "plugging" at the cryptal mouth resulting in the formation of a cyst or a cryptal abscess.

The rarity of cystic formations is remarkable when we bear in mind that these products of degeneration are claimed to be more frequent with advancing age. They were present more frequently in the connective tissue, mostly in the capsule, sometimes in the septa, but they occurred in the lymphatic tissue also. Only two of them were of the type described by Liveriero[22] as typical for advanced age; that is, walled in by squamous epithelium, containing partly amorphous and partly cellular elements, and showing a lack of reaction in the adjacent structures as a sign of degeneration. One of these two was found in a disorganized tonsil, the other in a fully developed one. The other cysts were found in almost equal number in fully developed and in markedly or maximally disorganized tonsils.

Similar distribution of the osseous and cartilaginous inclusions in the capsule and septa was seen. According to Hellman[14] they should be present more frequently in older persons, where they

originate from embryonic remnants as well as from ossifications, the latter being characteristic of cicatricial atrophy. All stages were represented; intramembranous and intercartilaginous ossification, the latter being best shown in partly cartilaginous, partly osseous, continuous structures. Once a piece of hyaline cartilage was found in the areolar tissue bordered by three lobules of salivary glands. Several times bone or pieces of bone and cartilage formed a chain immediately behind the lymphatic tissue, like parts of a calcified capsule. Once the lymphatic tissue around the bottom of a crypt contained unorganized osteoid tissue of low calcium content. It was impossible to correlate these findings with the process of involution, especially since one third of the bone and cartilage findings were present in tonsils without signs of regression. As the rest were found in disintegrated organs, it can be said that the tendency to build cartilage or bone is not diminished with advancing disorganization. Bone and cartilage, an almost typical part of the tonsil, remain simply the same characteristic feature of this organ up to advanced stages of atrophy.

Regarding the theories of bone and cartilage formation, no new viewpoints result from these findings. The osteoid tissue in a crypt bottom remains unique in the sense that the building of a calcified sheath is accomplished here not in the capsule, but in the lymphatic tissue. This condition may be a regressive metamorphosis of the connective tissue, as are the chains forming part of a bony retrotonsillar capsule, whereas the more isolated pieces may be developed from fetal rests.

The inclusions in the crypts were present as long as the crypts were closed enough to hold them in their lumina. According to Sponholz,[26] the highest incidence of their occurrence is reached between the ages of 20 and 30. This was verified in the sense that they are evacuated later with progressing eversion of the crypts.

The connective tissue does not display any qualitative changes which would be characteristic of an involutionary stage. As against the conception of Minear, Arey and Milton,[23] according to whom atrophy of the lymphoid tissue and compensatory formation of fibrous tissue accompany the degeneration of the crypt system, it has to be emphasized that lymphatic and connective tissue disappear in proportion to each other, even if individual differences are present. Schwarz[24] claimed that according to hereditary factors, these individual trends will be recognized in the regressing organ too. Sometimes larger bundles of connective tissue presented transparency and homogeneity.

Goodale[12] found that in retrograde metamorphosis of the tonsils, the endothelial cells of the follicles are seen first to diminish in number, while the lymphoid cells persist longer. In our experiments with tissue culture,[16] the final colliquation of the explanted tonsil has shown the following characteristics: the nuclei of the cells in the septal connective tissue were the first to lose their staining properties; the same phenomenon was observed in the lymphoid cells of the parenchyma considerably later. All these phenomena are but manifestations of the original type of the tonsil, as are the two different types af atrophy demonstrated by Goodale.[12]

It is claimed that a more conspicuous appearance of elastic fibers in the connective tissue accompanies advancing age. Tables 7 and 8 betray no such tendency. The rich elastic capsule around newly formed bone and cartilage can be explained in two ways. Either these pieces act as irritating foreign bodies and the fibers represent a reaction against them, or locally richly distributed elastic elements promote the formation of such inclusions, especially of the intramembranous type.

Elastic fibers help to outline the capsule and are present in almost all instances, according to Tables 3 and 4. They surround even small remnants and mark clearly the area of disintegration. How and when they disappear totally and become indistinguishable between the layers of the smooth wall of the empty tonsillar fossa cannot be followed with the help of operative material.

Tonsils obtained in this way will not permit one to judge pigment-deposits, especially in the hemolytic zone, as they may be blurred by even a small amount of bleeding from the operation.

The presence of a capsule to the very end of the period of involution is an important feature from the viewpoint of the operation. The plane of cleavage remains intact for a smooth enucleation. The intervention would be much more difficult if it were necessary to remove the remnants one by one.

Next to these general considerations, attention has to be directed toward the vessels of the capsule. Brunner[4] has pointed out their histological peculiarities. The arteries show cushion formations. The veins are present in two groups; a lateral one furnished with a muscular layer, and a medial one without muscles. The easily closing arteries, together with the veins of the described types, can bring forth rapid changes in the grade of the blood supply and may play a role in the fluctuating appearance of the secondary nodule. The same thing will occur at the height of the development, as in the

involution. As this set-up is the only one which might offer an explanation for fluctuations in the size of the organ, it has to be examined more closely.

Strawinsky[27] described thicker parts in the intima in the form of cushions in the arteries of the umbilical cord. These were found later in the bronchi, in the prostate, the kidney and the thyroid. Von Ebner[7] saw them in vessels from 1 mm. down to 0.2 mm. in caliber in the arteries of the penis, and Kiss[19] examined them in detail in his studies on the mechanism of erection. As Kiss described, one sees ridges most frequently at the points of branching; these are in certain cases 8 to 10 times as long as broad. Von Ebner thought that with simultaneous contraction of the circular muscle in the arterial wall, the longitudinal muscle fibers contained in the cushions or ridges would effect a perfect closure, while with relaxation the whole structure would hardly protrude from the level of the wall. Kiss made careful calculations, indicating that the difference in the lumina can hardly be narrowed down in this way more than one-sixth to one-fourth of the original width. These structures were found only in adults.

Examination of our own material presented the following: Elevations in the arteries can be found not with absolute regularity but quite frequently. The capsule of one tonsil may show them in abundance, another may be searched in vain for these structures. When they were present at all several of them were found. The interior of the cushion (in cross section) or the crista (in longitudinal section) always contained muscle. The elastic membrane beneath the intima was sometimes divided so as to build a sheath for these cushions.

Very conspicuous is the conglomerate grouping of the arteries and veins. The convolutes are usually seen immediately behind the lymphatic tissue in the most medial layers of the capsule. One group of vessels contained several protruding ridges or, in cross section, cushions. Where the artery is seen in a longitudinal section, several cristae may be present (Fig. 6). With the exception of three tonsils this picture was seen only when there was a conspicuous or extreme grade of disintegration. So far this was the only qualitative finding in the capsule to mark a definite involution. Brunner saw the cushions very infrequently in children but more often in adults.

The blocking of the blood supply momentarily plays an important role when there are temporary changes in the size of the sponge-like organ or in the closing of the vessels in the course of an

enucleation. It may result in definite atrophy of the organ if present over a longer period of time. The arteries and veins are present in many instances in the shape of conglomerates. We would call this disposition a plexus cavernosus retrotonsillaris. The arteries of the plexus are then provided with cushions or ridges. Figs. 5, 6, 7 show clearly how both factors cooperate in a highly efficient way.

A certain grade of endarteritis obliterans is sometimes present. It may be considered as physiological in an organ which ceases to function, as claimed by Boyd.[3] Diminished nutrition is the immediate result.

Thromboses in larger veins were not infrequently seen. Without perivascular infiltration no pathological importance could be ascribed to these findings. When they are present, one has to consider that with lymphatic cells in abundance in the vicinity, round cell infiltrations, as signs of a possible inflammatory reaction, can be judged only with great circumspection.

The epithelium of the everted crypt probably disappears rapidly in the further course of the involution. Krompecher and Némai[20] saw fatty degeneration of the superficial layer of the cryptal epithelium as a consequence of a lack of oxygen in the depth of the crypt; it was less marked on the everted surface in aging tonsils as this surface is more exposed to the air. According to Berggren and Hellman[2] general parakeratosis is more frequent in youth; later it is found only locally. Barnes[1] called this tendency a mild form of pharyngomycosis or hyperkeratosis tonsillaris. In our material local parakeratosis was counterbalanced by the opposite phenomenon—swelling and desquamation—which produced epithelial debris in the crypts.

Finally, the outstanding tissue of the tonsils must be discussed: the basal lymphatic parenchyma with its differentiations, the secondary nodules. Many kinds of involutional signs in it have been described by different authors. Barnes[1] found a correlation between the activity of the transmigration of lymph cells through the epithelium and advancing age. Smith[25] distinguished variations in activity, size and number of the germinal centers according to age. Berggren and Hellman[2] judged the grade of involution according to the content of connective tissue. They stated that in case one takes the amount of tissue in the secondary nodules as a sign of activity, then at any age one may find tonsils with a high-grade functional ability. They further mentioned that in older persons the connective tissue may decrease by shrinking. They also found larger

secondary nodules in children but there was no difference in their number in young or old. In still older persons they noted an increase of the dark zone.

The present material did not substantiate any of these findings. The size of the secondary nodules was within normal limits. Hellman[14] mentions a diameter hardly more than 1 mm., usually about .5 mm., as against the above recorded average of 561 microns. Heiberg's[13] figures for the largest diameter are between 200 and 1500 microns.

Berggren and Hellman[2] found the secondary nodules to be very resistant, disappearing only with the complete disintegration of the entire organ. Gaetano[10] saw them well differentiated in all stages of inanition (in lymph nodes).

According to our material, the earliest activity of the tonsils, that of sending white blood cells through the epithelium, is the last to disappear. Stöhr's old conception that lymphocytes pass through the epithelium, even in fetal life, with no differentiation into secondary nodules was confirmed by Foerster.[9] The latter put the first appearance of follicles several months after birth; Ehrich[8] found them in the fifth to the sixth month. At this period the transmigration has been going on for a long time. Thus the first and the last phases of the tonsillar life cycle demonstrate the fact that reticulation of the epithelium is a function which can be accomplished by the cells of the lymphatic tissue of the parenchyma without their ever having been differentiated into secondary nodules. Many tonsils in the material discussed in this paper showed long stretches of completely reticulated epithelium, in which the transmigration was accomplished by a simple underlying block of lymphatic tissue, without crypts and without formation of secondary nodules.

Thus the whole process of transportation of lymphocytes from their place of accumulation in the dark zones of the secondary nodules to the mouth of the crypt or, in the absence of this structure, to the free surface is exactly the same in the disorganized as in the fully developed tonsil. Fig. 4 shows this activity in a final remnant of the lymphoepithelial organ. Wessel's[28] finding of a diminishing number of outwandering lymphocytes in the phase of involution could not be verified. As for the lymphocytes themselves, Cowdry[5] points out that no change in their adult form betrays any individual senescence while the cells are circulating.

In the basal reticulum itself, there are sometimes seen free reticular spaces, invariably in the vicinity of the hemolytic zone;

that is, the boundary between the lymphatic tissue and the capsule. The emergence of such spaces in this location may have given the basis for the concept that atrophy of the tonsil begins in the neighborhood of the capsule and spreads toward the free surface. It was taken for granted that the free meshes represent fatty degeneration, especially since lymph nodes, retaining even in later life their original size, may be transformed into a mass of fat. But these reticular spaces are simply those which have been evacuated by the lymphocytes. This can be reproduced in tissue cultures of tonsils.[17] It is not easy to explain why in involution this phenomenon always takes place near the hemolytic zone. The ten tonsils in which such findings were most conspicuous were without exception in advanced stages of disorganization. Constancy of location and occurrence in disorganized tonsils excludes the possibility that the empty spaces are produced artificially in the course of histological preparation. A tentative explanation may be furnished by assuming that with fewer lymphocytes elsewhere the reserve near the capsule is mobilized; here the production could go on with greater intensity because of the higher vascularity of this region. Undoubtedly further observation and studies with fat-staining are needed to clear up this question.

The empty spaces themselves, like the pictures which show staining of the fibers of the basal reticulum, demonstrate that there is no enlargement of the "windows" and no decrease of the whole fibrillary network as is postulated by Hueck[15] for the aging mesenchymal reticulum. These structures are similar in construction and activity in the fully developed as well as in the totally disorganized tonsil (Fig. 3).

Much additional detailed work will be needed to clear up the understanding of the process of involution in the tonsils. More staining methods should be applied, especially for the demonstration of fat. Specimens taken at operation in older age groups should be examined carefully in every instance to get new information. Possible differences in the actions of tonsils of different age groups should be investigated. The role of the vessel system in the capsule should be followed up in serial sections.

SUMMARY

Involution in the palatine tonsils is an atrophic process, more precisely a numerical atrophy. Cells and tissues decrease in number without degeneration, and show no signs of change to any lower or functionally less active form before their final disappearance.

The capsule remains well outlined behind even the last remnant of the organ and thereby offers a plane of cleavage. Thus, enucleation in old age is a comparatively simple procedure. Bone, cartilage, and cyst formations are neither more nor less than they are in younger age groups. Septa and submucous connective tissue do not undergo any conspicuous hyalinization or transformation into elastic elements. There is no "senile fibrosis" in the tonsil.

Connective and lymphatic components disappear in equal proportion and the preponderance of one or the other corresponds merely to constitutional types observable at any age (parenchymatous or fibrous tonsil).

The epithelium shows shifting as the cryptal lining comes to cover the free surface after eversion. Even in this changed location the cryptal epithelium maintains its characteristically high grade of reticulation and remains sharply demarcated from the original epithelium of the free surface. The crypts contain their inclusions as long as they are not transformed from deep channels into open, shallow depressions.

The lymphatic parenchyma maintains, until its final disappearance, the function which was the first to appear in prenatal life, that of sending lymphocytes and leukocytes through the epithelium with reticulation of the latter. Cellular and basal-reticular constituents remain unchanged in form and activity, even in those last isolated blocks of lymphatic tissue which have already lost their differentiation into secondary nodules and their contact with any cryptlike structure.

Scarcity of thromboses in veins and of perivascular infiltrations in the retrotonsillar space accounts for the infrequency of local reactions around the receding tonsil. At the same time, with parenchymal activity unchanged, focal reactions appear at distant parts of the body, as they do at any age.

The grade of recession is generally the same on both sides. In the vast majority of the involuted group, tonsils may be found in all stages of development in the sixth decade. They demonstrate a tonsillar life cycle which is apparently independent of that of the organism as a whole. The impulse to start the involution is as unexplained in the tonsil as in any other organ. To attribute it to physiological aging or to describe it as a result of pathological processes remains pure speculation.

The double vascular mechanism of the retrotonsillar cavernous plexus with ridge-like structures in its arteries may be responsible for temporary changes in size at any age as well as for the definite closing off of the blood supply, which results in the final atrophy of the organ.

Acknowledgment is made to Prof. Benjamin Spector, Tufts College Medical School and Prof. LeRoy A. Schall, Harvard Medical School, for permission to use the laboratories of the Department of Anatomy, Tufts College Medical School, and of the Massachusetts Eye and Ear Infirmary.

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XXXV

DELAYED OSSIFICATION OF THE TEMPORAL BONE

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The following case deserves interest from both the theoretical and the practical points of view. Therefore, this exhaustive description seems justified.

REPORT OF A CASE

L. W., a five-months-old, well-nourished and well-developed white female child, was admitted to the Research and Educational Hospital, University of Illinois, on February 12, 1941. Her admission diagnosis was acute suppurative meningitis. According to the history given by the mother, the birth of the child was normal at full term. Her growth and development were normal. She was the youngest of three children. The family history was without importance. There was no deafness, no fragility of bones and no blue sclera in the family. The child was well until Christmas, 1940, when she took cold. This developed after visiting another family whose child had pneumonia and died within 24 hours. About the middle of January, 1941, the child developed an acute left purulent otitis media and a right postauricular swelling. By Feb. 5, the otorrhea on the left ceased spontaneously, but the swelling on the right became reddened. There was also a swelling on the right side of her face.

On Feb. 6, a right antrotomy was performed. Following the operation there was general improvement but the temperature still remained around 101° F. On Feb. 11, the mother thought the child suddenly became paralyzed as she was lying stiff in bed with her eyes staring straight ahead. She was this way for an hour with labored breathing. On Feb. 12, upon admission to the Research and Educational Hospital, her spinal fluid appeared cloudy and yielded pneumococci type 18. She was put on sulfapyridine and glucose intravenously.

Feb. 14, the left tympanic membrane appeared reddened. This was incised but no pus was found. The right external auditory canal was filled with cerumen and pus. The postauricular wound on the right had not healed and contained granulation tissue. The antrum mastoideum was well opened. The dura of the middle fossa was exposed and seemed to be under pressure. The dura was covered with granulation tissue. No operative procedure was recommended.

Feb. 13, because of failure of the child to respond satisfactorily to chemotherapy, a revision of the right mastoid cavity and a left antrotomy were performed.

On the right there was a small amount of granulation tissue covering the bony sinus plate. The plate was removed and the normal sinus wall was exposed. A puncture of the sinus yielded blood. The dura of the middle fossa was reddened and it was under pressure. The supero-posterior angle of the pyramid was removed. On the left there was no pus either in the mastoid or in the antrum. A small amount of granulation tissue was found in the antrum. The bone was soft.

Feb. 19, the child died. At autopsy an acute suppurative meningitis and a toxic nephrosis were found.

MACROSCOPIC AND MICROSCOPIC EXAMINATIONS

The skull and brain. On the right the dura was slightly thickened and the fontanelle was not bulging. There were no signs of increased intracranial pressure. The entire surface of the brain was covered with a thick yellowish plastic exudate. This exudate extended to the base of the brain. Almost all of the superficial cerebral veins were thrombosed and only faintly visible on account of the exudate, presenting a reddish-yellow, marble-like appearance. The pus extended well between the hemispheres. The ventricles were not involved.

The microscopic examination showed that the leptomeninges over the cerebrum and the cerebellum were filled with myriads of cocci, polymorphonuclear cells and debris. The underlying cortex was congested. There was superficial perivascular infiltration but the brain tissue was largely free from infection.

The temporal bones. The macroscopic examination showed the left drum membrane to be absent and the middle ear partly destroyed due to the removal of the temporal bones from the body. The right temporal bone was well preserved. No abnormality was recognized.

The microscopic examination showed that the mucous membrane was thickened from edema rather than from infiltration (Figs. 1, 2 and 3). The subepithelial connective tissue showed a marked thickening in the lining of the tympanic membrane and of the antrum. There was no inflammatory reaction. The epithelium was cylindrical and was well preserved. The hyperplastic mucous membrane presented a wavy appearance, filling the recesses of both windows and to a great extent, the cavity of the middle ear. Within the diminished middle ear cavity, there was serofibrinous exudate and occasionally pus. In the tegmen tympani there were many dehiscences filled with connective tissue and capillaries. However, no path of infection could be seen. There were dehiscences on the floor of the middle ear; these were filled with connective tissue. The jugular bulb was normal. The stapes appeared normal; its inferior part was fixed

by adhesion to the promontory. The stapedioincus articulation was also normal. In the short process of the malleus, islands of calcified cartilage were present (Fig. 3).

In the head of the malleus and in the entire body of the incus were many marrow spaces. A large marrow space excavated the long process of the incus and extended to the periosteum at several places so that there was no bone in these areas (Fig. 3). The marrow spaces contained loose connective tissue, capillaries and a varying amount of lymphatic cells. These lymphoid cells were not so numerous as to designate them as lymphoid marrow. The margins of these marrow spaces were either aplastic or showed the presence of osteoblasts. In some sections these osteoblasts laid down a fine seam of osteoid substance. Osteoclasts were not present.

The membranous inner ear. Both the right and left internal auditory meatuses contained much pus which extended toward the tractus forminosus but not into the modioli. This pus also invaded the cochlea and the facial nerves within the internal meatus. The cochlear canal presented a moderate ectasis. Reisner's membrane showed folds at several places. Corti's organ was preserved and normal. There was serous exudate in the endolymphatic and perilymphatic spaces of the cochlea. The blood vessels of the modiolus were dilated. The sacculus was dilated and contained serous exudate. The macula sacculi was normal. The cochlear aqueduct contained pus on the left. The veins of the aqueduct were greatly dilated. The utricle and semicircular canals were normal.

The ductus reuniens was slightly dilated and contained serous exudate. On the right there was a spot of atypical epithelium (Fig. 4). This area consisted of an accumulation of connective tissue which contained several dilated capillaries, but no nerves. The connective tissue of this atypical spot had no relation with the connective tissue supporting the wall of the ductus. It was separated from the latter by the basilar membrane. Toward the lumen of the ductus the pillow of connective tissue was covered by a layer of cubical cells. These were taller than those of the rest of the wall. There were no sensorial cells, no otolithic membrane and no otoliths. In the mesial and lateral walls of the right utricle there were spots of atypical epithelium. These were bud-like thickenings of connective tissue. In the inferior utricular sinus on the lateral and mesial walls there appeared such an atypical epithelial spot of particular size. This consisted of loose connective tissue, containing capillaries but no nerves. The area was covered by epithelium which had undergone cystic degeneration. At the base of the crista of the posterior canal there were cysts within the

Fig. 1.—Vertical section through the right temporal bone. *E*, external auditory canal; *D*, drum; *I*, incus; *S*, head of the styloid process; *F*, facial nerve; *HC*, horizontal semicircular canal; *SC*, inferior semicircular canal; *FC*, superior semicircular canal; *C*, calcified connective tissue.

epithelial layer. These cysts were covered by a plate or cuboidal epithelium and contained a fluid which stained deeply with hematoxylin.

The periosteal capsule of the cochlea and the vestibule. In the region of the cochlea and the vestibule, the periosteal capsule was well developed at the anterior and posterior surfaces of the pyramids. The width of the periosteal capsule was due to a large marrow space originating in the area of the attic above and extending to the hypotympanum below. The periosteal capsule surrounded the cochlea like an arch, and extended toward the superior and inferior walls of the internal meatus.

A large marrow space was present within the periosteal layer, but not between the periosteal and endochondral capsules. This marrow space was much better developed on the right than on the left. It contained lymphatic marrow with many enlarged capillaries. The marrow space also contained several spicules of lamellar bone. At several places these were covered by a blue "grenzscheide" (line of arrested growth). They formed no actual network. These bony

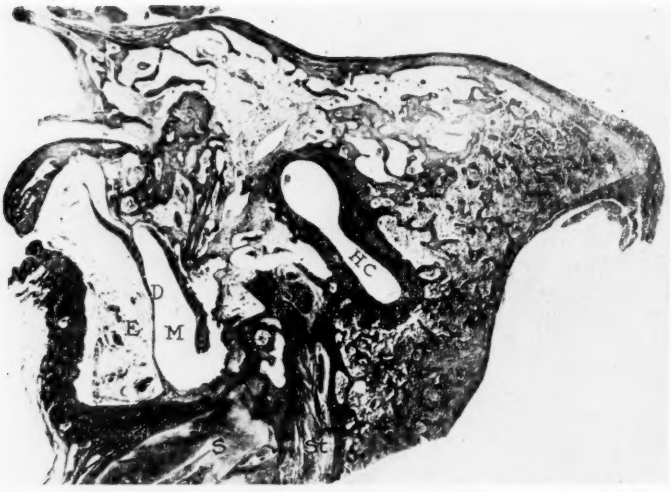

Fig. 2.—Vertical section through the right temporal bone. *D*, drum; *E*, external auditory canal; *M*, middle ear; S, head of the styloid process; *St*, stapedius muscle; *HC*, horizontal semicircular canal.

spicules connected the two layers of the periosteal capsules at several places. A number of these bony spicules contained a core of calcified connective tissue. The margins of the marrow space were entirely aplastic in that there were neither osteoblasts nor osteoclasts. Toward the attic and the hypotympanum the lymphatic marrow gradually changed into a loose connective tissue. At several points the marrow space was invaded by the epithelium of the mucous membrane of the middle ear. The epithelium formed several cysts but did not go on to the formation of pneumatic cells, although there was a slight metaplastic osteogenesis within the connective tissue.

A layer of periosteal bone separated the marrow space from the dura. Another layer separated the marrow space from the endochondral capsule. These may be called the internal and external periosteal layers respectively. Both layers consisted of woven bone with many osteocytes, some of which showed canaliculi. There was no abnormal increase in the number of osteocytes. Within the periosteal bone there were numerous capillaries and small marrow spaces. Haversian systems were absent. The marrow spaces contained capillaries and a loose connective tissue. The margins of these spaces were

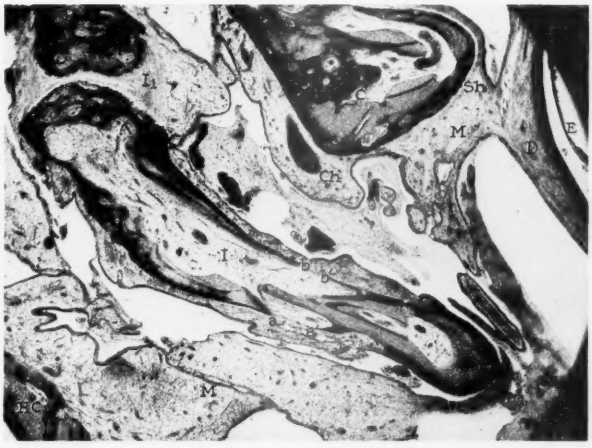

Fig. 3.—Section through the horizontal semicircular canal (*HC*); *I*, incus; *Sb*, short process of the malleus; *D*, drum; *E*, external auditory canal on the right side. *M*, mucous membrane of middle ear; *I*, body of incus; *CH*, chorda tympani; *C*, island of calicified cartilage within the short process of malleus. In *a* - a^1 and *b* - b^1 the periosteal bone is absent.

either aplastic or covered with osteoblasts with or without a fine seam of osteoid substance. Those with osteoid substance were more frequent. There were no osteoclasts.

In the internal layer of the periosteal bone, toward the dura, there were many dehiscences. In the inferior wall of the internal meatus the periosteal bone contained small spots of connective tissue, presenting metaplastic osteogenesis.

The endochondral capsule of the cochlea and the vestibule. The endochondral capsule surrounding the cochlea was thin. But it was much thicker in the fundus of the internal meatus and in the area of the vestibule. Within the capsule there were a great number of calcified interglobular spaces. The whole thickness of the endochondral layer had a bluish color in contrast to the periosteal layer which stained better with eosin. The endochondral capsule contained many small marrow spaces, corresponding to the age of the child. The marrow spaces in front of the oval window and in the promontorium appeared to be larger than in infants of similar age. The marrow spaces contained dilated blood vessels and a loose connective tissue

Fig. 4.—Section through the ductus reuniens, (*Dr*) on the right side. *Ex*, exudate; *I.D.*, insertion of the secondary drum; *E.atyp.*, atypical epithelial spot within the ductus reuniens presented under higher magnification in picture b which shows: *e*, the epithelial wall of the ductus reuniens; *c*, capillaries; e^1, the epithelium of the atypical epithelial spot; *co*, connective tissue; *B*, basement membrane.

with many lymphocytes and plasma cells. There was neither osteogenesis nor osteoclasis. The margins were covered with a blue "grenzscheide". Into these marrow spaces entered small spicules of bone and cartilage. These were also covered with a "grenzscheide". There was only a small amount of bone within the endochondral layer, but that which was present was normal in structure. In several places the bone contained a great amount of osteocytes with or without canaliculi.

There was a distinct line of demarcation between the periosteal and the endochondral layers. As mentioned above, both layers differed in their color. Frequently there was an appositional arrest-line between these layers. The line consisted of a cement-like substance which stained deeply with hematoxylin or of a calcified ground substance of cartilage. Interglobular spaces never extended into the periosteal layer but ended abruptly where the two layers met. In the area of the promontorium the endochondral marrow spaces laid down a thin layer of lamellar bone without interglobular spaces. An appositional arrest-line separated this layer of lamellar bone from the periosteal bone of the promontorium. The endosteal capsule of the inner ear was normal.

The area of the semicircular canals. According to M. Meyer,[10] under normal circumstances the periosteal bone penetrates deeply into the pyramid between the endochondral bone which surrounds the

tube-like membranous semicircular canals. That finding can be explained in the following way: The connective tissue of the fossa subarcuata and the retroarcuata (Eckert-Moebius) pushes forward between the semicircular canals. In so doing periosteal tissue is displaced into the depth of the pyramid. From the displaced periosteal tissue, periosteal bone is formed. Thus the presence of periosteal bone between the semicircular canals is easily understood. Meyer also states that in the newborn the convexity of the anterior and the posterior semicircular canals is separated from the dura only by an endosteal layer. In a later period of life periosteal bone is laid down on endosteal bone without intercalation of endochondral bone. Thus on the convexity of the vertical semicircular canals enchondral bone is frequently missing. In the case reported the convexity of the posterior semicircular canal (Fig. 1) and the crus commune was covered with an extremely thin layer of endosteal bone. The convexity of the anterior semicircular canal was covered with endosteal and periosteal layers of bone. It is probably inaccurate to call this finding pathological in a child five months of age.

The subarcuate fossa was large and consisted of delicate connective tissue. The parts of the semicircular canals which did not encroach on the cranial fossa were covered by bony tubes which consisted only of endosteal and endochondral bone (Figs. 1 and 2). The latter was sclerotic and contained numerous interglobular spaces and osteocytes, but only small marrow spaces. A normal temporal bone at this age contains periosteal bone between the semicircular canals. In the case reported the periosteal bone was replaced by connective tissue of the fossa subarcuata and by the large periosteal marrow space. The latter contained several bony spicules, but less than in a normal infant of four and a half months of age. Thus the picture presented was that of bony semicircular canals, consisting of endosteal and endochondral bones, being embedded in connective tissue and lymphatic marrow (Figs. 1 and 2).

Below the superior angle of the pyramid, there was an accumulation of calcified connective tissue which was attached to the dura of the posterior fossa (Fig. 1). Within the calcified connective tissue there were spicules of woven bone. In the left temporal bone the findings were less marked. However, the left semicircular canals were not completely dissected.

The area of the oval and round windows. Both the round and oval windows were normal. In front of the oval window there was much endochondral bone which was covered by a thin layer of periosteal bone. Within the endochondral bone there were many small

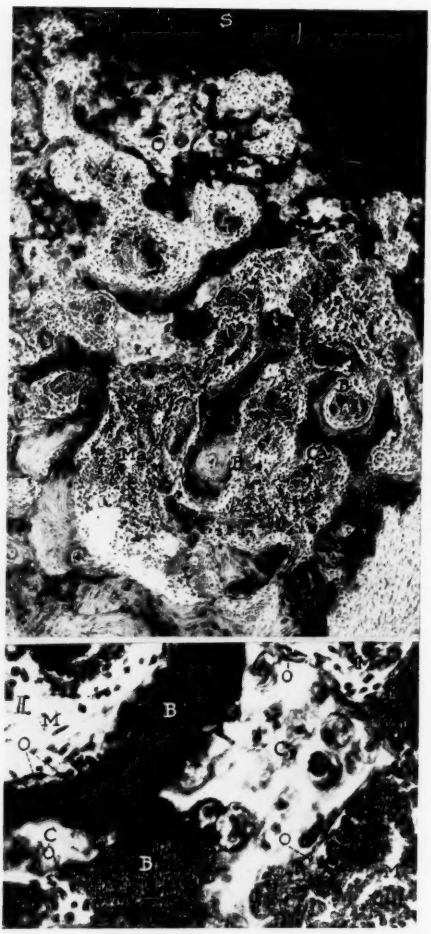

Fig. 5.—The upper section represents the ossifications zone (*O*) in the head of the styloid process on the right side. **S**, calcified cartilage; *B*, spicules of newly formed bone in the center of the marrow space (*Ma*), laid down on spicules of calcified cartilage (*CA*). *x* is represented under higher magnification in the lower section where *M* indicates the marrow space, *B* primitive bone with large osteocysts, *O* osteoblasts with osteoid substance, *C* - *C*[1] distended and degenerated cartilage cells embedded in bony ground substance.

marrow spaces which in several places fused to a large marrow space. In these marrow spaces there were capillaries and connective tissue which in several places joined with the subepithelial connective tissue of the mucous membrane of the middle ear. There was neither osteogenesis nor osteoclasis within the marrow spaces. In front of the fissura ante fenestram there was a large piece of cartilage which extended from the bony facial canal to the basilar coil of the cochlea. The cartilage was surrounded by endochondral bone and consisted of partly calcified and partly noncalcified cartilage. It failed to show signs of ossification. The fissura ante fenestram divided the cartilage into a larger superior and a smaller inferior portion. Both of these disappeared when the annular ligament made its appearance. The cartilage on the right was much less developed than that on the left.

COMMENT

Three findings are of great interest: (1) the spots of atypical epithelium in the membranous inner ear, (2) the changes of the bony capsule of the inner ear and of the ossicles; and (3) the meningitis which invaded the inner ear along the internal auditory meatus and the cochlear aqueduct.

1. There were bud-like thickenings of the lateral and mesial walls of the utricle, cysts at the base of the inferior crista and a thickening of the wall of the ductus reuniens which had the appearance of a macula. The thickening of the wall of the utricle is frequently observed. This is particularly true in the wall of the sinus utricularis inferior. There was a unanimity of opinion in the past that the latter thickening should be considered as a remnant of a macula neglecta. We are in perfect accord with Alexander[1] and Fischer,[2] who do not consider this point important but whose chief concern is centered on whether or not sensorial cells and nerves are present within these thickenings. Like the majority of cases already reported, in the present case both sensorial cells and nerves were absent but distinct blood vessels were observed within the thickenings.

The fact that Fischer[2] found the walls of the vestibule thickened in human embryos indicates a persistence of fetal formations. Brunner[3] found them in human embryos 18 mm. in length. They were either solid or hollow. The hollow ones simulated glandular structures. These thickenings are uneven in appearance and are always found in the mesial wall of the sacculus which is later endowed with the macula sacculi. The thickenings in the saccular wall gradually disappear with the growth of the embryo. As far as we know, in the newborn the wall of the sacculus shows no thickenings.

It is difficult to understand why the ear models of Streeter do not show these thickenings.

Fischer[2] has shown that the wall of the utricle of the human embryos also contains thickenings. This was confirmed by Brunner.[3] The latter author has shown that these utricular thickenings are inconstant, while the saccular thickenings are present in all human embryos at certain stages of their development. It is not clearly understood why, in postfetal life, these thickenings always disappear from the sacculus while they may persist in the utricle.

The cysts at the base of the sagittal crista must also be considered as persistent fetal formations. Fischer[2] found similar cysts in the superior and horizontal cristæ of human embryos. It will be recalled that these cysts concern the epithelium of the crista which is lying above the basilar membrane. Brunner[4] found similar cysts in the supporting tissue of the crista, that is to say, below the basement membrane of the epithelium, probably originating from a different source. Of particular interest is the thickening of the wall of the ductus reuniens (Fig. 4). This is seldom seen in the human. A number of years ago Alexander[5] noted a similar thickening of the wall of the ductus reuniens in embryos of guinea pigs which had the appearance of a macula. He considered it as a formation analogous to the papilla lagenae of amphibia and fish.

All these observations indicate a disturbed development of the membranous inner ear in our case.

2. The changes of the bony capsule of the inner ear are best observed by comparing it with a normal temporal bone of a similar age. We are indebted to Dr. John R. Lindsay, Chicago University, who placed at our disposal the serial sections of the temporal bone of an infant at the age of four and a half months.

The changes in the present case are more marked on the right than on the left, the left temporal bone having been incompletely sectioned. The endosteal layer in both inner ears fails to show any abnormality. The endochondral layer is not yet completed, corresponding to the age of the infant. However, it does seem that there is a greater number of calcified and noncalcified interglobular spaces and of marrow spaces than in normal infants of this age.

More conspicuous are the findings in the periosteal layer, the greater portion of which consists of a large marrow space containing hyperemic capillaries and lymphoid marrow. In the region of the cochlea the marrow space is separated toward the endochondral layer as well as toward the dura by a relatively thin layer of periosteal

bone. Neither osteogenesis nor osteoclasis was observed along the margin of the marrow space. There are a few spicules of bone within the marrow. These spicules do not form a network and some of them contain a core of calcified connective tissue — a picture usually seen in an infant of two or three months of age. A normal five-months-old child should have much smaller marrow spaces with bony spicules which are thicker and more numerous regardless of the degree of pneumatization as Dr. Lindsay's specimens showed.

Most striking are the findings in the area of the semicircular canals. In a normal child at this age the area between the bony semicircular canals is largely filled with periosteal bone. In the case presented this was substituted largely by lymphoid marrow. Furthermore, in normal infants the small marrow spaces within the periosteal layer contain not only connective tissue and capillaries but also osteoblasts, osteoid substance and occasionally osteoclasts. In the present case the periosteal marrow contains osteoblasts occasionally, osteoid substance rarely, and osteoclasts never. Similar changes in osseous development are found in the malleus and the incus in which the marrow spaces without osteoclasts are larger than in a normal infant.

Summarizing it may be stated that there is a delayed osseous formation which chiefly concerns the periosteal capsule and in lesser degree the endochondral capsule. This is much more marked in the area of the semicircular canals than in the region of the cochlea. So far as bone has formed it has a normal structure.

In the light of these observations one might be led to assume that these findings are variations of ossification, since the processes of pneumatization and ossification are subject to considerable variation. In a more careful analysis there are findings in the present case which do not permit this concept of a simple variation in development. First, considering the age of the child, the delay in bony development exceeds the physiological limitation. Second the large focus of calcified connective tissue below the superior angle of the right pyramid (Fig. 1) and the partial absence of periosteal bone in the long process of the right incus (Fig. 3) can scarcely be deemed as a simple variation. Third, a variation of endochondral ossification is recognized by variation in bony deposition and cartilaginous resorption. These processes of deposition and resorption occur hand in hand. The speed with which this is accomplished is subject to considerable variation but always follows the normal pattern. In the case presented, this process of ossification is irregular and incomplete. It fails to follow the normal patterns as noted in the right styloid process. In a normal

infant at this age the head of the styloid process consists of cartilage which presents the changes preliminary to ossification. In our case the cartilaginous cells fail to form regular rows. The cartilage is more calcified. Far more important are the changes in the ossification zone (Fig. 5), in that much calcified cartilage escaped destruction by advancing marrow. Thus the amount of bone laid down by the primary marrow is definitely diminished. Away from the zone of ossification there is an increase in the number of bony spicules within the marrow space. The majority of these spicules contain cartilaginous ground substance, even cartilage cells which are not influenced during the process of ossification. Occasionally swollen cartilaginous cells with a degenerated nucleus are noted within these bony spicules (Fig. 5).

This type of ossification presents features that are indicative of an abnormal process: the destruction of cartilage definitely prevails over the formation of bony spicules engulfing the cartilage in the center of the marrow space.

In summarizing we are led to believe that the findings in our case cannot be considered as simple variations of ossification but rather a retardation of ossification caused by pathological conditions. These pathological conditions are well known although not too clearly understood. They are called osteogenesis imperfecta. There are two types. One is called osteogenesis inperfecta Vrolik or the congenital form. This is supposed to be due to a congenital inferiority of the osteoblasts. The other is called osteogenesis imperfecta (adult form) or osteopsathyrosis idiopathica of Lobstein. It has not been fully established whether the two types are two distinct clinical manifestations of a single morbid entity or two different diseases. Most of the pathologists are inclined to the first concept (Kramer[6]). J. Fischer[7] was the first to describe the changes in the temporal bone in a verified case of osteogenesis imperfecta congenita. His observations were later confirmed by Nager,[8] Weber,[9] Meyer,[10] Nager and Meyer[11] and by Altmann.[12] These changes consist of a delayed and abnormal formation of bone in the periosteal and endochondral layers, while the bone itself has a normal structure. All agree that the changes are most pronounced in the area of the semicircular canals and that there is a varying degree of ossification in each individual case.

Obviously our case presents all the findings of cases of osteogenesis imperfecta congenita except one important feature. All writers describe changes in the rest of the skeleton as being characteristic of osteogenesis imperfecta congenita. The skeleton of our patient was not examined either by x-ray or by microscope. However, so marked are the changes of the skeleton in this disease with its tend-

ency to multiple fractures, bending of long bones, and a persistence of the membranous vault of the skull that it could not have escaped the notice of either clinician or pathologist. Thus our case does not come under a typical case of osteogenesis imperfecta congenita.

Let us now consider osteogenesis imperfecta tarda or osteopsathyrosis. The temporal bones in such cases were examined by Ruttin,[13, 14] Gimplinger[17] and Altmann.[12] Ruttin's case was in a 24-year-old man in whom the bony capsule of the labyrinth was normal except for an otosclerosis in the region of the oval window. A similar observation was made by Gimplinger in a 54-year-old woman. Altmann's case was in a 17-months-old girl who died from a widespread tuberculosis. In her skeleton there was but a small amount of periosteal and endochondral bone. There was an old fracture of the left femur but there were no blue scleræ. In her temporal bone the capsule of the labyrinth did not present any anomalies except a marked delay of ossification in the mastoid, in the temporal squama and in the tip of the petrous bone. It is a question whether or not the case of Altmann presents an actual osteogenesis imperfecta, as the fragility of the bones could have been due to the generalized tuberculosis. At any rate it is generally agreed that in osteopsathyrosis an otosclerosis is the only definite finding that occurs in the bony labyrinth, although otosclerosis is more frequently observed without fragility of long bones.

To properly classify our own case is extremely difficult since there is no way to determine the appearance of the labyrinthine capsules of the patients reported by Ruttin[13] and Altmann[12] at five months of age. Nor do we know what the appearance of the bony labyrinth and the remainder of the skeleton of our patient could have been had she attained the age of 17 months or 24 years. The task of a correct classification is made doubly difficult since there were no blue scleræ, no blue drum membranes and no significant history of tendency to fracture in the long bones. The great amount of atypical epithelial spots in the membranous inner ear points to the tentative diagnosis of an osteogenesis imperfecta congenita. There are malformations not only in the mesenchymal but also in the ectodermal structures. However, no definite diagnosis can be made from these findings alone. What can be said is as follows: In the bony capsule of the labyrinth and in the ossicles of the middle ear of a five-month-old girl there were found characteristic changes of osteogenesis imperfecta but she presented no classical symptoms as far as her skeleton was concerned. Whether these findings indicate the earliest signs of an osteopsathyrosis is difficult to state. Then, too, whether this child would have presented blue scleræ, brittle bones, and even otosclerosis

in later life can not be definitely predicted inasmuch as the rest of the skeleton was not examined. This element of uncertainty led us to name the title of the present communication "Delayed Ossification of the Temporal Bone" and not "Osteogenesis Imperfecta Localisata of the Temporal Bone."

3. The child died from meningitis due to pneumococci, type 18. The source of the meningitis could not be ascertained since the lungs and the middle ears showed no definite pathology. However, there are several features that point to meningitis of otitic origin. First, the history of bilateral acute otitis media; second, the presence of exudate within the right middle ear; and third, free vascular communications between the mucous membrane of the middle ear and the dura through the tegmen tympani and the antrum. The fact that the blood vessels and accompanying connective tissue did not show any signs of inflammation does not speak against the otitic origin of meningitis. It is characteristic for the infections of the middle ear due to pneumococcus to heal spontaneously, while the intracranial complication makes its appearance.

In the presence of frank suppurative meningitis the operative procedure on the mastoid becomes useless as it was in this instance. The best measure at our command is an adequate and well selected chemotherapy. Unfortunately, the child failed to respond to chemotherapy. As a last measure the mastoid operation was performed, more to satisfy the relatives than from actual indication.

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XXXVI

AN IMPROVED METHOD OF NARROWING THE NOSE

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Since Joseph[1] did the first nasoplastic operation in 1898 for reducing the size of the nose by removing a hump and shortening and narrowing the tip, there has been a notable growth in the realization of the importance of nasoplastic surgery. This thesis deals with a modification of the method of narrowing the nose; in all other respects the operation is similar.

This operation is not only important to cosmetics, but also to psychology and economics, because there may be serious disturbances of the emotional life and difficulty in securing employment.[2]

These plastic operations concern only the external nose and not the intracranial nasal cavities.

Anatomy. The framework of the nose is made up of both bone and cartilage. The bony portion consists of the two nasal bones, one on either side of the midline. These are supported by the perpendicular plate of the ethmoid, which forms the bony septum, and by the frontal processes of the superior maxillae.

The cartilaginous portion consists of the two upper lateral cartilages, which are joined with the perpendicular septal cartilage, two free lower lateral cartilages, and several small accessory cartilages (Fig. 1).

The two nasal bones, which make the bridge of the nose, are in contact at their upper edges with the frontal bone. Their lower edges are attached to the upper lateral cartilages and their lateral edges are attached to the frontal processes of the superior maxillae. The medial edges meet with each other in the midline.

From the services of Dr. George M. Coates of the University of Pennsylvania Hospital; Dr. Benjamin H. Shuster and Dr. Seth Brumm of the St. Luke's Medical Center, Philadelphia.

Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania, in fulfilment of the requirements for the degree of master of Medical Science for graduate work in Otorhinolaryngology.

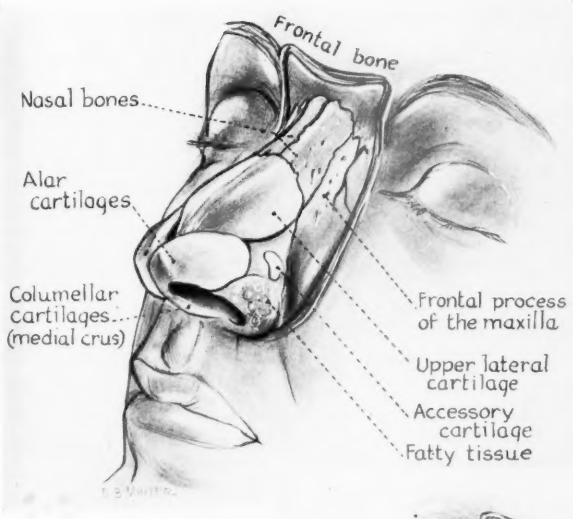
Frontal bone
Nasal bones
Alar cartilages
Columellar cartilages (medial crus)
Frontal process of the maxilla
Upper lateral cartilage
Accessory cartilage
Fatty tissue

Fig. 1.

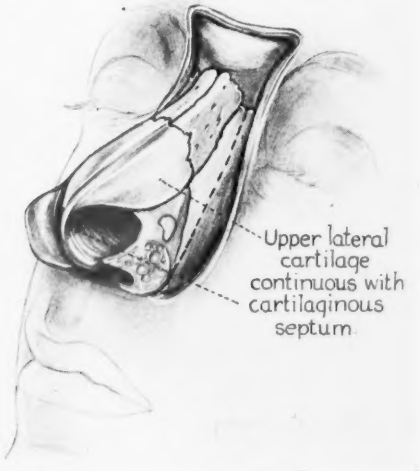
Upper lateral cartilage continuous with cartilaginous septum

Fig. 2.

The upper third of the bony portion of the septum is made up of the perpendicular plate of the ethmoid. It includes also the vomer, the spine of the frontal bone, the rostrum of the sphenoid, and the crests of the nasal, maxillary and palatal bones.

The vomer forms the postero-inferior part of the nasal septum. Its anterior edge is grooved to receive the septal cartilage. The most anterior extremity of this edge is in immediate contact with the incisive crest of the maxilla. This contact is of particular importance surgically, since the lower segment of the septal cartilage which supports the nasal tip is inserted here. If the adjacent nasal spine has been sacrificed in a submucous operation, the tip of the nose may become undesirably lowered due to the sinking of the nasal dorsum.

The upper lateral cartilages, which form the middle third of the nose, are contiguous to the lower borders of the nasal bones. Each of the lower lateral cartilages consists of a lateral and a medial crus, the latter of which surrounds the nostrils. The nasal septum, which forms the partition between the nasal fossae and which supports the dorsum of the nose, is made up of septal cartilage, the vomeronasal cartilage, and the medial crura of the lower lateral cartilages.

This septal cartilage makes up the larger part of the medial partition of the external nose. It is quadrilateral in shape and is continuous anteriorly with the upper lateral cartilages, which spread away from it in winglike fashion (Fig. 2). The lower septal edge is short and has a fibrous tissue attachment to the lower lateral cartilages.[3, 4]

Preliminary Examination. A thorough study of all the patients is made before operation. It is of particular importance for the success of plastic surgery that the subject be in good physical condition. First, a careful physical examination is given. If there is evidence of a mental or an emotional disturbance, the patient is sent to a psychiatrist.

Records of special examinations are made, as follows:

1. Wassermann test
2. Complete blood count
3. Urinalysis
4. Bleeding and coagulation time
5. Still photographs
   (a) Direct profile
   (b) Smiling profile to judge the length of the upper lip

(c) Front face (erect) to show deviations and other abnormalities

(d) Front face with head tilted backward to show the shape of the nostrils

6. Profilometer measurement
7. Motion pictures (Kodachrome)
8. Facial mask (plaster of Paris).

If any physical disorder is present or if any of the laboratory tests indicate a pathological condition, operation is postponed until such time as the patient's condition is again normal.

The patient's photographs are placed on a music stand at the head of the operating table, where they can be studied from time to time as the operation proceeds.

The masks, which are made of each patient according to Berson's method,[5] are constructed in the usual manner. With these masks, the necessary facial measurements, with which the ideal nose should conform, can be determined. Two planes of the face are concerned: the front view and the direct profile. The masks are placed on a table which stands at the head of, and on a level with, the operating table. This allows the operator to make comparison with the field of operation from time to time.

Instruments. The necessary instruments include a speculum, a pronged retractor, a Bard-Parker knife (No. 11), a double-edged knife curved on the flat, a button-end knife, an angulated knife, a periosteal elevator, Joseph's right and left bayonet saws, Joseph's right and left right-angled saws, a straight scissors, nasal rasps, tissue forceps, mosquito hemostat forceps, dural hooks, needles, a needle-holder, silk, a recording syringe with various sized needles, a small chisel and a mallet, Stent's modelling compound.

Instead of the customary bevel-edged chisel, the writer has found more satisfactory for his own method of operating the fusiform-shaped chisel with wedge-shaped edge. It will be considered later in describing the operation.

Preoperative Measures. Sulfathiazole and calcium lactate, ten grains twice daily, are given for three days before operation.

In preparing the preoperative field, very strict asepsis must be sought. About an hour before the operation is to begin, the hairs

about the nostrils are removed with fine-pointed scissors. This should be done with particular care so that there shall be no injury to the nasal mucosa. The nasal vestibules are then scrubbed with cotton applicators wet with green soap and thoroughly flushed with sterile water in order to rid the field of any foreign substance. Care should be exercised not to injure the nasal mucosa. After this cleansing has been thoroughly done, ether is applied on a swab and the field thoroughly dried.

For preliminary anesthesia, each nasal fossa is packed with a strip of half-inch gauze moistened with equal parts of 10 per cent cocaine and 1:1000 adrenalin. Not more than 30 minims are necessary. This packing is allowed to remain in place three minutes.

In these operations, as elsewhere, the patient's individual idiosyncrasy to drugs should be kept in mind, and any to which there is sensitivity should be carefully avoided.

THE OPERATION

The patient's head is closely wrapped in sterile towels, leaving only the face exposed, and he is then ready for the operating table. The head and shoulders should be somewhat elevated to facilitate operative movements and to help in keeping nasal congestion and hemorrhage at a minimum.

Two sterile towels are laid under the head. The upper one is wrapped about the head closely and secured with towel clips. A drop of sterile castor oil is put in each eye, after which the entire face and nasal cavities are scrubbed with green soap and water for a number of minutes and generously rinsed with sterile water. Finally, the face is dried with sterile gauze followed by a rub with alcohol and then with ether.

Before anesthesia of the external nose is begun, the upper part of each nostril is packed with sterile gauze to prevent blood from escaping into the nasopharynx. The patient's eyes and mouth are then covered with strips of gauze.

External Anesthesia. Local anesthesia is the method of choice in these operations, as it leaves the field free for the operator. However, if it is indicated, as it may be in some cases, a general anesthetic can be given and should be administered by the endotracheal method.

With the direct method of local anesthesia of the nasal pyramid, three intradermal wheals are raised: one at the junction of the bony

and the cartilaginous dorsa and one on the side of each ala. Through these wheals the afferent nerves of the operative field are blocked. In order to increase the anesthetic area, the injected field may be gently massaged.

Not more than 8-10 cc. of 1% novocain are usually required to secure the desired narcosis, but if this amount should prove inadequate, the needle may be left in place while the syringe is refilled. This method prevents unnecessary added injury to the nasal mucosa and other tissues.

Infiltration of the dorsum of the nose is first done by inserting a long needle intranasally at a point between the upper and lower lateral cartilages. The needle is then advanced submucously to the glabella and then withdrawn slowly with gradual expulsion of the contained solution. The lateral wall is then infiltrated by inserting the needle at the vestibular base of the ala and carrying it forward to the nasofrontal suture, with expulsion of the contained solution as the needle is slowly withdrawn. The needle is again inserted at the same point and the solution is introduced under the periosteum. As the final step, a shorter needle is used to infiltrate the base of the nose by radial injections around its circumference.

Occasionally, it is desirable to enhance the effect of local anesthesia. To do this, the sphenopalatine ganglion may be injected by the external route. A line drawn downward from the outer canthus of the eye and a line drawn parallel to the nasolabial junction will intersect at the point at which the ganglion is to be injected. At the point of intersection, a fine 7 cm. needle is thrust backward, upward and medialward until the needle reaches the pterygomaxillary fissure, which is the region of the ganglion. Five cc. of novocain to which one drop of adrenalin has been added in the proportion of one drop to the dram, are injected, with resulting deep anesthesia.

Operative Method. With the left thumb and forefinger the operator lifts the tip of the nose and introduces a knife or double-edged scissors into the vestibule between the upper and lower lateral cartilages, and the aponeurosis connecting the two cartilages is cut through. A pair of scissors is introduced through this incision and passed under the skin over the upper lateral cartilage until the nasal bone is reached. The blades of the scissors are opened and the skin lateral to the nasofacial fold and medial to the nasal dorsum is undermined. This same procedure is then repeated on the opposite side.

After the skin is detached from the underlying structures, the periosteum is raised through the intranasal incisions.

When the soft parts have been separated, a button-end scalpel is introduced through the left incision, passed over the dorsum and downward until it appears through the nasal incision on the opposite side, where it is turned at right angles.

Bony Operation. If the hump to be removed is small, a rasp may be sufficient to remove the excess of bone. For this method, the skin and the periosteum are elevated through the intranasal incision and a nasal rasp is introduced and manipulated with an up-and-down movement. This reduces the hump to produce the desired nasal profile. Bone fragments should be removed with a dull curette or by means of a cotton applicator wet with hydrogen peroxide.

If the bony hump is large, separation of the periosteum from the bone is not advisable. To operate, the saw is introduced into the intranasal incision and placed against the bone on the exact line where the profile is to be reduced. External palpation will determine the correct position, as will also the position of the handle of the saw. The nasal bone and the perpendicular plate of the ethmoid are then cut through with a few up-and-down movements of the saw. For this part of the operation it is advisable to place the forefinger and thumb of the free hand on the skin over the saw, in order to guide its direction and keep it from slipping, and thus prevent injury to the overlying skin. In case the saw clogs, it must be removed and cleaned. The next step is to repeat the operation on the opposite side of the nose. If any fragments of the hump remain attached to the septum, the periosteum or the upper lateral cartilages, they can be separated with a blunt-end knife, introduced under the upper end of the bony hump and drawn down toward the tip of the nose. The hump can then be lifted out with strong forceps.

The necessity of cutting the hump on exactly the same lines on both sides should be emphasized. If this is not done, the dorsum will be irregular. Any lack of uniformity can be corrected by the use of a rasp.

The Flattened Bridge. However small the amount of bone removed from the bridge of the nose, a flattening results. To correct this flattening it is necessary to fracture the frontal processes of the maxillae and displace them toward the median line.

To fracture the frontal process of the maxilla, double-edged scissors or a knife is passed through the mucosa of the vestibule at the nasofacial junction and carried through the soft tissues until it reaches

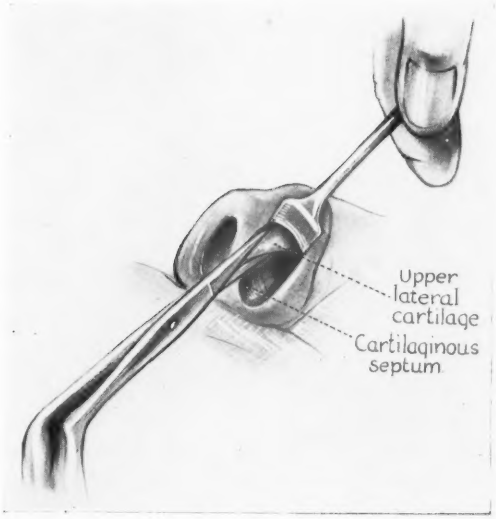

Fig. 3.

the pyriform opening. With a periosteal elevator, the periosteum is raised at the lower border of the pyriform opening up to the naso-frontal suture. The saw is then introduced under the periosteum and engaged in the nasofacial groove,[9] with its base at the most anterior margin of the pyriform opening and its apex at a point half way between the orbit and the glabella. The position of the saw should be controlled by two fingers of the free hand. About two-thirds of the thickness of the bone is cut through, special care being taken that the mucosa is not injured. This operation is repeated on the opposite side.[1, 6-8]

In the ordinary technique, the fracture to narrow the bridge is completed by turning the patient's head away from the surgeon, and with the thumb padded with gauze pressure is applied upon the bone fragment of the maxillary process. The pressure required for this, though often described as "gentle," is in reality considerable and is accompanied by an audible cracking of the bone, which is disturbing to some patients. The fracture, too, takes place in the wrong direction. To avoid this situation, the writer has introduced a means of completing this fracture, which has been used in a series of 75 cases and has proven in every way preferable.

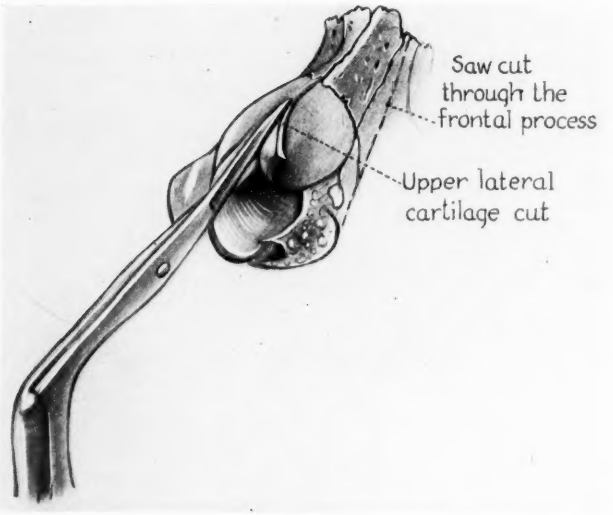

Fig. 4.

Author's Technique. The lateral cartilages are separated from the septum (Figs. 3 and 4). After the required fragment of the maxillary process has been partially separated with the saw, instead of using pressure to complete the fracture, a slender bi-convex chisel 8 inches long and 5/8 inches in width at its greatest convexity is engaged in the first saw-line with a few light taps of the mallet. The handle of the chisel is then directed towards the ear, and by easy leverage the fracture is made and the bone fragment is displaced outward. After removal of the chisel, the detached fragment is easily pushed downward and into place with the thumb. This technique insures a cleaner break and avoids any disturbance to the patient.

The shape of this chisel has the particular advantage of greater accuracy in determining the direction of the fracture line (Fig. 5).

Narrowing the Lobule. Removing the hump and shortening the nose will give the lobule too wide an appearance and this must be corrected. An incision is made just below the inferior edge of the lower lateral cartilage inside the vestibule. A pair of double-edged scissors is introduced into the incision with the instruments lying between the exterior skin and the cartilage. With the blades opened, the

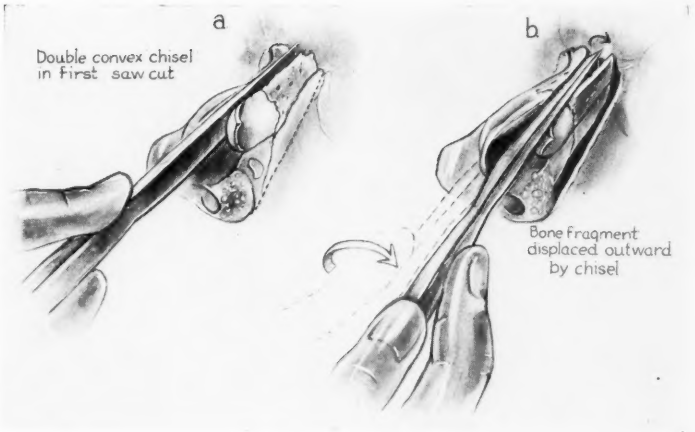

Fig. 5.

tissues are separated laterally to the posterior edge of the alar crus and medially to the septum. The cartilage is freed in the same manner from the skin lining the vestibule, except that the scissors now lie between the cartilage and the vestibular skin. The liberated cartilage is withdrawn from the vestibule with a single-hook retractor, and a strip of cartilage is resected from the angle, its width and length being governed by the size of the reduction to be made. The separated parts of the lower cartilage will fall into the midline, and the reduced width of the tip of the nose will correspond to the width of the fragments of cartilage which are removed.

Postoperative Procedures. Following the operation, all packs are removed from the nose, which is then compressed from above downward to force out any possible blood clots. The face is washed off with a moist sponge and the nostrils are repacked with kephrine hydrochloride gauze. This is a 2¾ inch sterile, selvage gauze permeated with 1:1000 adrenalin and dried. It controls postoperative capillary bleeding. The gauze is partly coated with sulfonamide ointment, which is used to reduce the possibility of infection.

To prevent edema and the effusion of fluid into the tissues about the eyes and the surrounding part of the face, the writer applies a right pressure bandage and a gauze packing over the eyes. Without these this region may become discolored following the operation.

Except for inspection, dressings and bandages are not changed until the fifth or sixth day, according to the method of Aufricht.[10]

Original drawings from the operating room and anatomical specimens.

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PLATE I

PLATE II

XXXVII

THE USE OF RADIUM IN CONDUCTION DEAFNESS

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It has been conclusively proven by many investigators that obstruction of the lumen of the eustachian tube is the most common cause of conduction deafness. Valsalva[7], in 1707, described self-inflation of the eustachian tube as a means of cleaning mucus from the ear and as an aid in treating deafness. Mayer[5], in 1868, first recognized the important role the adenoids play in eustachian tube obstruction and advised their removal as an aid in relieving otitis media. Henle[4], in 1868, was the first to describe the microscopic anatomy of the eustachian tube. Politzer[6], in 1863, was one of the first to introduce an air douche for inflation of the eustachian tube and the middle ear. However, as the years passed and knowledge regarding the anatomy of the eustachian tube become gradually disseminated, it remained for Crowe and Baylor[3] and Burnam[2] to give us a satisfactory therapeutic method for eliminating the all-important nasopharyngeal lymphoid tissue which is the chief etiological factor in eustachian tube obstruction.

The nasopharyngeal lymphoid tissue lies directly between the orifices of the eustachian tubes. When this tissue is hypertrophied, as is the case in practically all children and many adults, it causes partial and complete obstruction of the eustachian tubes. The function of the eustachian tube is to ventilate the middle ear and the pneumatized spaces in the petrous portion of the temporal bone and the mastoid. If the tube is obstructed, the air in the middle ear is absorbed, the mucous membrane becomes congested and serum and mucus are secreted to fill the space. If this condition remains unrelieved, the tenacious myxomatous mucus and ultimate fibrotic changes in the middle ear interfere with the mobility of the ossicles. A gradual loss of hearing insidiously develops, affecting first the high tones with gradual progression toward the lower end of the scale.

For years otolaryngologists conscientiously removed the nasopharyngeal lymphoid tissue in children with curette or adenotome, but failed to realize that this specialized tissue was an integral part

of the mucous membrane and could not possibly be completely eradicated unless the nasopharyngeal mucous membrane was removed in its entirety. If they removed the lymphoid tissue (as was done in the vast majority of cases) prior to puberty, the small remaining nodules would always undergo a compensatory hypertrophy. Thus investigators finally, in desperation, turned to a study of the effects of radium and roentgen rays on lymphoid tissue.

Akaiwa and Takeshima[1], in 1930, carried out experiments on the effects of irradiation of the lymph nodes found in the popliteal spaces of rabbits. Thirty minutes after exposure to irradiation the lymph follicles of these nodes were enlarged. After one hour the nuclei of many of the cells began to show disintegration, which reached its height in from two to six hours. From this time on marked phagocytosis was noted, the phagocytes taking up the chromatin particles of the decomposed nuclei. Because of this disintegration of lymphocytes there was a considerable decrease in the number of these cells in the lymph follicles after 48 hours.

Following the above phenomena there was a period of regeneration, so that after the seventh day the lymph node resumed its normal appearance except for an increase in the connective tissue. Macroscopically there was an increase in the size of the node after the first two hours following irradiation. There was then a gradual decrease in the size of the node, and in two weeks the node was smaller than normal. It thus follows that, if a satisfactory method could be obtained for exposure of the nasopharyngeal lymphoid tissue to the rays of radium, it could be easily and painlessly removed from its environment.

Crowe, Baylor and Burnam devised a nasopharyngeal applicator in which was held a glass capsule containing radon. Children whose eustachian tube orifices were obstructed by lymphoid tissue were given a two-gram, minute treatment at each eustachian tube orifice. A marked diminution in the size of the tissue was noted, and the hearing was vastly improved in those individuals who exhibited a loss of hearing.

With the foregoing facts in mind I have used this treatment in 45 carefully selected cases of conduction deafness. A large mass of lymphoid tissue was noted obstructing the orifices of the eustachian tubes in each case.

Instrument—The instrument used is slightly different from that used by Crowe, Baylor and Burnam. It consists of a copper wire 7 inches long which is attached to a platinum capsule 0.5 mm. in

thickness. The capsule is loaded with 50 mg. of radium. The platinum capsule and the wire are covered with a coating of gum rubber 0.7 mm. in thickness.

Method of Application—A one per cent cocaine solution is lightly sprayed into each inferior meatus. A cotton-tipped applicator moistened with three drops of ten per cent cocaine is then gently passed along the floor of each inferior meatus. After an interval of five minutes the radium applicator is inserted through the inferior meatus into the nasopharynx. A two-gram, minute treatment is given on each side. This treatment causes the patient no discomfort whatsoever, and can be given to children and adults alike. During the first week following the treatment there may be a slight subjective sensation of "stuffiness" or "fullness" in the nasopharynx, accompanied by a very moderate increase in the postnasal discharge. This is followed by a diminution in the amount of postnasal discharge, so that four to five weeks after treatment there is no noticeable postnasal discharge. Examination of the nasopharynx six weeks after treatment reveals a definite diminution in the size of the nasopharyngeal lymphoid tissue as well as a decrease in the amount of tenacious mucoid discharge.

Results—The results have been uniformly encouraging. Each patient has shown improvement in his ability to hear following the treatment, as shown by audiometric studies taken before and after treatment. These patients have been examined six weeks following treatment and reexamined after twelve weeks. Eleven patients had to have a second treatment and three patients had to have a third treatment.

CONCLUSIONS

1. Nasopharyngeal lymphoid tissue will undergo a compensatory hypertrophy, producing a large recurrent mass of adenoids and a "granular" pharyngitis, in almost every child whose adenoids have been removed prior to puberty.

2. Large masses of nasopharyngeal tissue which obstruct the eustachian tubes are definite etiological factors in conduction deafness.

3. Atrophic changes can be readily produced in this tissue by exposing it to the gamma rays of radium.

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XXXVIII

BRONCHIAL LAVAGE IN NONTUBERCULOUS INFECTIONS

HOWARD L. STITT, M.D.

CINCINNATI

Nontuberculous infections of the bronchial tree, though born of trivial and preventable causes, may assume pathological proportions sufficient to incapacitate the individual and to render him a burden to himself and his neighbor. The morale of the staunchest who may be a victim of purulent bronchitis, bronchorrhea or bronchiectasis with or without fetor is broken by long periods of distressing cough and expectoration of foul-smelling sputum. To bring these patients help has long been the purpose of the medical practitioner, but because even those most successful in their aim have as yet by no means accomplished all they might wish, any attempt to add to the approved schemes of treatment should at least be examined.

Kolb[1] and Ellis[2] in 1879 were probably the first to recognize bronchiectasis clinically and to suggest for it a specific line of treatment. From that time until now investigators have vacillated in their estimates of the value of treatment as obtained by essentially local medical and surgical means and by other means directed constitutionally.

The significance of clinical evidence of bronchial suppuration must not be misunderstood. Pus is discharged not only in bronchiectasis but in pulmonary abscess and purulent bronchitis. Roentgenologic examination helps in the recognition of abscess and bronchiectasis, as does the discovery of dilatation, cavities and pneumonic involvements situated along the bronchial tree. Accumulations of noninfected secretion should not be mistaken for lung abscess. The lateral views are often called for. Stereoscopic plates identify the types of dilatation (cylindrical, globular, and racemose). Iodized oils, first injected by Sicard and Forestier,[3] mark the size, form and extent of the dilatation. Through roentgenograms solitary and retro-

Presented as a Candidate's Thesis to the American Broncho-Esophagological Association, 1942.

cardiac enlargements and dilatations are frequently revealed which might otherwise have been overlooked.

Endobronchial examination is essential. The age of the lesions may thus be calculated. Young lesions are smooth and glistening, the mucous membrane is usually pale. Later they become granular and dull. Chronic lesions are smooth and fibrous, exhibiting granulations that frequently bleed. The endobronchial examination should determine the size, shape and configuration of the bronchial tree and the color and texture of its mucous membrane, also the amount, kind, source and character of the secretion. At such examination an uncontaminated specimen must be taken for pathologic and bacteriologic examination.

Since 1933 we have used for preliminary bronchoscopic examination a 7-40 modified Jackson bronchoscope[4] to catheterize the smaller bronchi from which one sees pus and granulation exuding. One can insert a #6 ureteral catheter through the special channel into the discharging bronchi from the bronchiectatic or abscessed cavity. By this process one can aspirate the pus with the assistance of the nurse, after the catheter is placed in position by visual direction. At this time irrigation with a slightly hypertonic saline solution and a medicament can be applied. We find this more effective than simply aspirating the main bronchus. Furthermore, we advise frequent bronchoscopic examination with this catheterizing bronchoscope, say three to four times within a year, to check any new development which might occur in the course of the treatment, such as excessive granulation, strictures, or excess purulent secretion. In the meantime bronchial lavage with the Coudé catheter is done. Endobronchial examinations, however, also have their limitation. Dilatation of the small bronchi cannot always be made out. Localized emphysema and peribronchial abscesses may be overlooked, though the bronchoscope does identify the particular branch which is the source of the pus.

Sicard and Forestier[3] offer iodized oils as a diagnostic aid and treatment. The opaque oil marks size, form and extension of dilatation. Solitary dilatation and retrocardiac dilatations are frequently revealed which might otherwise be overlooked.

For bronchial disinfection and immunization the use of suitable chemical and biological agents, recently advocated by Kolmer,[5] rests upon sound therapeutic principles, as it takes into consideration endobronchial drainage, the restoration of bronchial function, the destruction of infectious agents and the establishment and mobilization of local tissue defenses.

Bronchoscopic drainage as developed by C. Jackson, Tucker, Clerf,[6] Lukens, Moore, Funk[7] and others has definitely improved the treatment and prognosis of bronchiectasis and allied conditions by providing a means for removing collections of purulent secretions and making direct or topical applications of medicinal agents. The bronchoscope cannot always reach the smaller bronchi and it is not practical to make direct applications through it with the frequency demanded. Therefore, any method that permits such application with the minimum discomfort and expense to the patient and permits the removal of bronchial secretions and instillation of bactericidal and bacteriostatic agents, without tissue destruction, is to be welcomed as opening up a possible means of improving the treatment for these distressing infections.

C. and C. L. Jackson[8] state that, after bronchoscopic study of the pathologic conditions present and after clearing away the bronchial obstruction, it may be desirable to supplement the bronchoscopic examination with aspiration, suction, irrigation and medication with a catheter introduced through the larynx into the bronchi with or without a stylet.

It is worthy of record that the first advocate of intrabronchial medication was Green.[9] He was censured by his contemporaries. Bronchial lavage by catheter was advocated by Garcia Vicente[10] in Spain and thought to be a valuable adjunct to bronchoscopic treatment in suitable patients.

What we deem to be our contribution to the treatment of purulent nontuberculous affections of the bronchi and lungs by lavage and aspiration is this:

1. We have found a soft curved tip rubber catheter with the hole in the distal end #16 French (Coudé[11]) preferable to frequent lavage and aspiration with the bronchoscope.

2. We have found in most patients immediate altering of the symptoms following irrigations with a harmless slightly hypertonic salt solution, supplemented with antiseptics, proper diet, rest and hygienic surroundings.

3. In the attempt to reduce the effects of infection, we have found the best aid to be a slightly hypertonic salt solution because it is least harmful to the involved tissues themselves.

The instruments for such a lavage are: a soft rubber catheter #16 French (Coudé)[11] without stylet, a metal tongue blade, a head

mirror, an emesis basin, a 60-cc. metal ear syringe, a solution basin, an intratracheal syringe to administer the local anesthesia, and a small graduate for the subsequent medicament.

The technique is simple and is as follows: The patient is seated in an upright position; the nurse stands behind the patient, supporting his head; a 10 per cent larocaine solution[12] or a 2 per cent pontocaine solution in hypertonic saline (we employ only the Bledsoe-Fischer saline solution[13]) is then injected into the larynx and the subglottic area. After the local anesthesia has taken effect, the tongue is depressed with a tongue blade. The curved tip of the catheter is then introduced behind the epiglottis, pushed through the glottis into the trachea and down to the main carina. The tip is rotated by touch either to the right or to the left main bronchus. (An indicator to the direction of the tip may be added to the proximal end at first, but is not needed once you acquire the touch method. We advise placing the patient before a fluoroscope in the beginning to acquire the touch method). The nurse grasps the catheter with the thumb and forefinger. The patient holds the emesis basin. After some three or four experiences, the patient becomes tolerant of the catheter so it becomes unnecessary to anesthetize the larynx or the tracheobronchial tree. In unilaterally affected cases we insert the catheter by touch to the diseased side for lavage. In bilateral cases we first lavage one side, then the other, or hold the tip of the catheter just above the carina to allow the hypertonic salt solution to enter both main bronchi. At the conclusion of the lavage we introduce the medicament.

The proximal end of the catheter is attached to the metal syringe and 15 to 30 cc. of the hypertonic saline solution is slowly introduced into the bronchi coincident with each inspiration. By reason of the cough reflex and the peristaltic action of the bronchi, the solution, carrying with it the thick ropy secretion, is expelled around the catheter into the emesis basin. The action resembles vomiting. As much as 50 to 100 cc. of the solution may be used. Following the cleansing lavage any other medicament may be instilled. Very seldom is it necessary to use any additional local anesthesia to favor their retention.

The Bledsoe-Fischer hypertonic saline solution is a mixture of various salts, which, as in the case of Ringer's solution, are less harm-

ful to living tissue than any salt alone. It is prepared from the following mixture:

Sodium Chloride	263.7
Potassium Chloride	10.6
Calcium Chloride (dessicated)	21.0
Distilled water q. s.	1000 cc.

Twenty to 22 cc. of this concentrated mixture is diluted with 500 cc. of distilled water for intratracheal and bronchial use.

This solution contains the three salts which Ringer originally found so favorable for the maintenance of a physiologic activity of living tissue. It constitutes, therefore, a so-called physiologically balanced mixture. The concentration of the sodium chloride has been raised to a point where it will not only prevent swelling of the injured human cells, but somewhat above it. The concentration of the calcium chloride is raised to several times that in Ringer's solution in order to maintain the dehydrating effects of the various salts as long as possible. While monovalent salts, through their more rapid diffusion, affect the inflamed tissue first, the dehydrating effects of the calcium salts lasts longer, once this salt has become diffused into the tissue.

We found that the hypertonic Bledsoe-Fischer solution is in itself cleansing and will decrease and thin the bronchial secretion, eliminate the saprophytes, decrease the fetor and the bacterial flora until the patient has less secretion, a greater feeling of well-being and an increase in weight to the extent that he can be classed as symptomatically well. With the use of medicaments following the lavage more lasting results are obtained.

A germicide of choice for bronchial disinfection must be relatively nontoxic and should in a well-tolerated concentration be capable of effective antibacterial action without destroying the defensive mechanisms of the cells.

Many medicaments have been used for antiseptic or bacteriostatic ends. Yankauer[14] employed monochlorphenol and iodine; Moore[15] trinitrophenol and iodine. We have used both but with only fair results. Following irrigation with the Bledsoe-Fischer solution, we have found valuable a solution of 1.52 per cent pyridine, quinoline and isoquinoline in a special hydrocarbon oil (metrol oil). These are obtained from a double distilled oil of shale. This does not irritate the bronchial mucosa. The quinoline has an effective analgesic

action but does not inhibit the normal cough reflex. Following its use we have observed a great decrease in bacterial flora of the lung and a decrease in fetor. This solution is particularly effective in chronic cases which do not respond to lavage alone. Ten cc. of the oil is injected into the bronchi on inspiration and allowed to remain. We used this medication until 1933 with good results up to a certain point. We found then that merthiolate (aqueous), mercresin (aqueous) and metaphen (aqueous) reduced to a certain degree bacterial count and fetor. Lately we have used Ceepryn, a brand of cetylpyridinium chloride (aqueous), diluted with Bledsoe-Fischer hypertonic saline solution to make a 1:5000 solution. This has proved the most valuable germicide we have used so far and we now use Ceepryn exclusively. It is highly germicidal, has a decided low surface tension action and is nonirritating in the concentration used.

It has been suggested by many investigators that food rich in vitamins and minerals favors regeneration of normal epithelium in the endobronchial tree. An adequate amount of protein and inorganic material should be provided. More nearly native foods should be substituted for white wheat flour and cane sugar. The less pure salts (because they contain magnesium, calcium and iron) are superior to the refined grades of table salt.

"Biologicals" did not help our patients.

It has been established by Spink[16] and others that under certain circumstances there are distinct advantages in applying the sulfonamide compounds directly to the infected tissues. The method permits the application of higher concentrations of the drugs to a relatively small area. Our clinical experience has been limited to the use of sulfanilamide and sulfathiazole.

Since 1925 we have performed more than 8000 bronchial lavages associated in most instances with some kind of antiseptic treatment. They were performed on approximately 500 patients afflicted with chronic bronchitis, lung abscess (not peripheral) or bronchiectasis (unilateral or bilateral) essentially purulent in type. We have had three deaths which, however, we feel were not due to the treatment but to unrecognized myocardial complications. These patients had bronchiectasis.

We have refused to treat with bronchial lavage the diffuse suppurative pneumonitides, lobar and bronchial pneumonias, acute bronchitis and tuberculosis or patients afflicted with aneurysm, heart disease, hypertension, or angina pectoris.

Only occasionally do patients have a reaction following bronchial lavage such as a chill with rise in temperature (99° F to 101° F). In the course of four to six hours they have usually recovered. We believe that we have never disseminated infection.

Our method of procedure in all these patients begins with careful physical examination followed first by stereoscopic films then by films after iodized oil injections with the Coudé catheter. A few days after this a bronchoscopic examination is made to determine any other lesions complicating the condition.

We perform our bronchial lavages by employing a curved tip catheter, #16 Coudé. Its use makes hospitalization of most patients unnecessary and being more easily manipulated, its use is followed by less discomfort. Local anesthesia only is called for, which as the patient becomes accustomed to the procedure may be dispensed with, particularly in children. The curved tip of the catheter permits its introduction by touch alone into either the right or the left main bronchus.

The soft curved tip catheter (Coudé) lends itself to several other uses, such as the intrabronchial injection of iodized oils, the aspiration of secretion from localized areas and the obtaining of uncontaminated specimens from each main bronchus.

The ambulatory type of treatment is of pronounced economic value to the patient because more treatments may be given him at less cost. Patients do not fear the introduction of this catheter as they fear bronchoscopic procedure.

Repeated lavages with hypertonic salt solution (Bledsoe-Fischer) bring about an immediate cleansing of affected tissues. Better blood supply follows the cleansing effects of the slightly hypertonic salt solution so that this inhibits the continued existence of anaerobic and partial tension organisms found in the diseased tissues. Mere thinning of the secretions from the diseased areas enables nature better to carry off damaging products through ciliary action and cough.

In March, 1941, we began injecting a suspension of 20 grains of sulfanilamide crystals in a tragacanth base in nine selected patients ill with bronchiectasis, either unilateral or bilateral. All were infected with hemolytic streptococcus and staphylococcus and had been ill from four to fifteen years. Previous bronchial lavage with Bledsoe-Fischer hypertonic saline solution cleared the fetor and bacterial flora to a certain extent. Antiseptic medicaments such as oil of shale, merthiolate, mercresin, ceepryn chloride and biologic agents have been used in their treatment. All of these reduced the bacterial flora and

fetor to an even greater extent, so that the patients improved as evidenced by lessened discharge, greater comfort, gain in body weight, and a resumption of work and social position in life, but they were never entirely free of a productive purulent cough.

After 10 to 15 injections of sulfanilamide in these patients over a period of six weeks, uncontaminated specimens still showed hemolytic streptococcus and staphylococcus. We then decided to use 5 per cent sulfathiazole sodium sesquihydrate having a pH of 9.0. Twelve cubic centimeters were injected into each main bronchus following lavage with hypertonic saline solution. After approximately 15 intrabronchial injections of sodium sesquihydrate solution had been given to each patient over a period of six weeks the purulent discharge was perceptibly lessened. At this time we added sulfathiazole 7½ to 15 grains by mouth every three hours until a blood concentration of 3.0 to 4.5 mg. per 100 cc. was obtained. This treatment was continued on each patient for about six weeks.

Since August, 1941, two of these patients who had been ill for the past ten years have had no purulent expectoration. They have gained in weight and resumed their vocations. Bronchial lavage and subsequent medication have been stopped for three months. There are four other patients in this group who, we feel, will soon be free of expectoration. There have been no blood dyscrasias since we supplemented the sulfathiazole medication with a ferrated liver extract and vitamin B_1 and B_2.

We have had five patients with lung abscess (central) cured by bronchial lavage with hypertonic saline solution alone.

In approximately three hundred patients with chronic purulent bronchitis, bronchial lavage, followed in most instances by antiseptics, completed their cure. All of the patients with chronic purulent bronchitis complicated with bronchial asthma were relieved after a few bronchial lavages.

Since in bronchiectasis the cavities can never be eliminated or narrowed to normal limits except by surgery, complete anatomical cures cannot be obtained. However, we can conservatively state that of approximately 150 patients symptomatic cures have been obtained in 30 per cent; 50 per cent have received symptomatic relief and 20 per cent were unimproved.

Therefore, we are of the sincere opinion that these percentages justify this treatment by bronchial lavage with its subsequent medication.

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Clinical Notes

XXXIX

NASAL MYIASIS

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Nasal myiasis, the presence of the larva of the fly in the nasal cavities may be classified clinically in three main divisions:

1) Cavity and wound myiasis, in which the larvae invade natural or artificial cavities in the body, such as the nose, sinuses, and surface wounds.

2) Cutaneous myiasis, in which the larvae live in or under the skin.

3) Intestinal myiasis, in which the larvae live in the intestinal tract.

There have been described three chief groups of flies that may invade man. They are: the Calliphoridae and Sarcophagidae or blow flies, and the Oestridae, or warble and bot flies. Most forms of the first two groups live in decaying meat and are found only occasionally as parasites. When they do invade man, the flies feed on and destroy with their powerful jaws both the soft tissues and bone. If they are not removed, serious and even fatal results may ensue.

The screw-worm, or the larval stage of Chrysomyia macellaria, is of this type and belongs to the blow fly family. Any fly may invade and oviposit in the nasal cavities of man but the screw-worm is by far the most frequent invader. The Oestridae, however, normally pass their larval stage as parasites and they seldom cause the death of the host, unless present in unusual numbers or situated in some unusual location.

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Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania, in partial fulfilment of the requirements for the degree of Master of Medical Science (M. Sc. (Med.)) for graduate work in Otolaryngology.

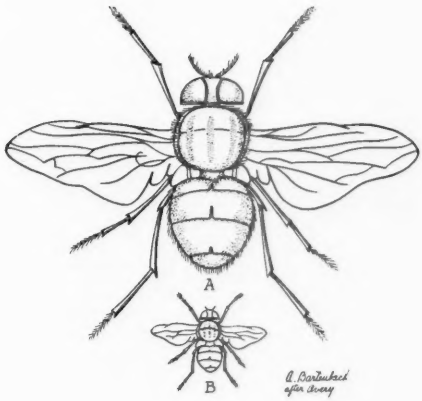

Fig. 1.—Chrysomyia macellaria (screw-worm fly). *A*, three times actual size; *B*, *actual size.*

The females of the screw-worm fly, Chrysomyia macellaria, deposit their eggs in batches of 50 to 200. The eggs are normally laid on dead animals, though the flies may oviposit on all sorts of sores, wounds, ulcers, in the nasal and aural cavities, and in other natural openings of the body. The eggs hatch in eight to ten hours and the larval growth is extremely rapid. Under favorable conditions mature larvae may appear as early as three days, though normally five to six days are required. The larvae live on the serum, the blood, and the decaying or living tissues into which they bore by means of their powerful mouth hooks. When mature the larvae leave the wound or body cavity and drop to the ground where they bury themselves in loose earth or debris. Pupation takes place within two to three days and the entire life cycle from egg to egg usually occupies less than two weeks.

The screw-worm is a pest in tropical and subtropical America. In the United States, it is most prevalent in the South and Southwest. The adult fly looks like a blue bottle fly but has three black stripes on the thorax. There is some question as to whether the female fly deposits eggs or larvae, but it is believed that when living animals are selected the method of infestation is by eggs. The larvae which hatch out from the several hundred eggs deposited wander in all directions.

If they are in the nasal cavities they may, and often do, eat their way into the sinuses causing considerable injury and suffering. The mature larvae are about two-thirds of an inch in length and have twelve rings of minute spines, which make them resemble a screw.

Many flies instinctively deposit their eggs or larvae where there is the odor of decaying or suppurating animal matter, and the screw-worm especially is a serious pest of domestic and game animals in the South and Southwest. The adult flies are attracted to the decaying and suppurating wounds of animals, resulting from fighting, dehorning, castrating, and branding, and oviposition frequently occurs in these wounds. The same smell of decomposition is present in the discharges of many natural orifices of man, especially if there is an accompanying suppuration, and a very common finding in the tropics is that of fly larvae in the middle ear in which there is present a chronic suppurating discharge, and in the nasal cavity in which a chronic foul smelling discharge is found .

In the south and southwestern parts of the United States, the favorite place of attack on man is the nostrils, but apparently only if a foul-smelling nasal discharge is present. The act of oviposition in man usually takes place while the person is asleep or in a drunken stupor, either in an unscreened room or in the open fields.

SYMPTOMS

Within 24 to 48 hours after invasion of the nose by the fly, there develops an irritation of the affected side which is evidenced by a tickling sensation, accompanied by frequent sneezing attacks. This is often followed within a few hours by a partial or complete unilateral nasal obstruction. The sneezing is followed within 24 hours by a serosanguineous discharge, which rapidly changes to a foul, purulent discharge. Swelling of the affected side of the face ensues at this time, together with considerable pain and an elevation of the temperature. The temperature may vary from a low-grade fever to a temperature of 103° F. to 105° F. in the neglected cases.

On the third or fourth day after invasion, many of these patients will complain of a very annoying sensation of movement or activity within the nose. Although none of the patients in the cases reported were of a particularly sensitive type, this sensation of movement, with the one exception of pain, was their most troublesome symptom. Eye signs, such as lachrymation, photophobia, and injection of the conjunctivae are frequently present. Nasal bleeding is almost always present to some extent, and severe epistaxis occasionally occurs, and

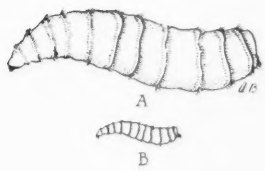

Fig. 2.—Chrysomyia macellaria (larva). *A*, three times actual size; *B*, actual size.

may occur, either early or late in the course of the disease. The patient consults a physician either because of sneezing, blowing, or spitting out one or more larvae or because of the increasing severity of the symptoms.

DIAGNOSIS

The diagnosis of this disease is usually very simple. The patient often comes to the physician complaining of maggots or worms in the nose, which he has previously sneezed or blown from his nasal cavities. This statement is quickly confirmed by anterior rhinoscopy, which reveals a foul, purulent, and slightly bloody discharge, containing numerous larvae that are almost always easily seen. In an early case, in which no larvae have been expelled, an inanimate foreign body must be considered in the differential diagnosis. In an early case there may be enough mucosal swelling so that the larvae are not immediately seen. However, the use of a shrinking solution will soon reduce the swelling and bring the maggots into view, and the diagnosis is made. In addition, the extensive tissue destruction present in all larvae infestation cases is not seen when an inanimate foreign body is the cause of the symptoms.

TREATMENT

The treatment is essentially that of quickly removing the etiological agent of the disease, together with appropriate supportive and local therapy. The etiological agents, the screw-worm larvae, are best removed by first stunning them with chloroform and then removing the larvae from the nasal cavity with a bayonet forceps. There are several ways in which the chloroform can be applied, and they are all effective:

1) The use of a chloroform spray, from an ordinary atomizer or spray bottle, instilled into the nasal cavity.

2) The use of cotton pledgets saturated with chloroform, which are inserted into the nostrils and left for several minutes.

3) The use of chloroform nasal douches in strengths ranging from 25 to 50 per cent.

One of these three forms of chloroform therapy together with mechanical removal of the larvae should be repeated two to three times daily, until all larvae are removed.

Supporting treatment is very valuable and should consist of relief of pain, heat to the involved area, adequate food intake, and a high fluid intake. In addition, adequate doses of a sulfonamide should be given to control complications if present, and to prevent them if not present. In two of the cases presented in this paper almost pure cultures of streptococcus hemolyticus were found in the nasal smears and cultures.

COMPLICATIONS

The most common complication is an acute paranasal sinusitis, and of the sinuses the maxillary are the ones most commonly involved. In many of these cases the sinusitis is an acute exacerbation of an already present chronic purulent sinusitis. The most dangerous complication is a meningitis, resulting from direct extension of the infection through the cribriform plate to the meninges. Epistaxis occurs frequently, and there have been cases reported in which it was severe enough to cause death. Bronchial and intestinal myiasis may rarely be complications of nasal myiasis. Necrosis and destruction of the nasal architecture is always extensive. The entire ethmoid labyrinth is often completely or partially exenturated, together with extensive destruction of the nasal septum, the turbinates, and portions of the lateral nasal walls.

REPORT OF CASES

CASE 1. The patient, a well-nourished man of 56 years, was admitted to the University of Kansas Hospitals on October 1, 1939, complaining of pain in the nose and face and a serosanguineous discharge from his left naris. The patient dates the onset of his illness to September 28, 1939, when he suddenly developed a severe pain over the left side of his nose and face. Simultaneously he developed a serosanguineous discharge from his left nostril. The pain had been intense for the past 24 hours.

On admission the patient was found to be moaning and complaining of severe pain in his face. He was extremely dirty in appearance and had a very foul odor which could be traced to his nose. On examination the mouth was found to be edentulous, and there was edema of the uvula and soft palate. Anterior rhinoscopy revealed a foul-smelling, serosanguineous discharge from the left nostril, and numerous maggots could be seen high in the left nasal cavity. His temperature on admission was 100.8° F., and his white blood count was 12,200. The Wassermann and Kahn tests were negative, and a roentgenogram of the sinuses revealed a chronic pansinusitis. The patient gave a history of nasal catarrh with a foul nasal discharge of 20 years' duration.

On October 1, 1939, the patient's left nostril was packed with chloroform-saturated cotton pledgets, which were removed in two minutes. Twenty maggots were then removed with a bayonet forceps. On October 2, a similar procedure was followed and ten maggots were removed. On October 3, five maggots were removed, and the patient still required morphine sulphate, grains one-sixth, every four hours for relief of pain. The bloody nasal discharge was still profuse. On October 4, no maggots were found and the nasal discharge was not purulent and only slightly blood-tinged. On October 5, three maggots were removed, but the patient was much improved and codein sulphate in small doses now controlled his pain. On October 6, seven dead maggots were expelled by blowing his nose; there was no pain, and the swelling of the face had completely subsided. The patient had no sulfonamide therapy and was discharged on October 10, 1939.

CASE 2. On September 15, 1940, a 20-year-old, white, male imbecile was admitted to the San Diego General Hospital with the complaints of maggots in the nose and recurrent, profuse nasal hemorrhages. The patient had suffered from chronic sinusitis accompanied by a foul odor for ten years, according to his parents.

On September 12, 1940, the patient began to have slight nasal bleeding, accompanied by sneezing attacks, both of which gradually increased in severity. On September 14, a physician was consulted, who instilled a mercury preparation in the nose, following which he removed 24 maggots by suction. The nostrils were then tightly packed with iodoform gauze to control hemorrhage. On September 14, the patient began to hemorrhage profusely from the postnasal region and he was sent to the San Diego General Hospital.

On admission the patient was comatose and had a slightly stiff neck. The Kernig and Babinski signs were negative. The anterior

nasal packing was removed and ten maggots were removed after the use of a chloroform spray. Because of excessive bleeding a postnasal tampon was inserted, together with anterior nasal packing. A tentative diagnosis of meningitis, secondary to maggot infestation, was made. Although bleeding was controlled, the patient gradually became weaker and expired within three hours of admission.

An autopsy was performed by the coroner, and he considered the immediate cause of death to be "ulceration of the nasal septum due to pansinusitis." General toxemia and larval infestation were listed as contributory factors. A postmortem examination of the brain was not performed.

CASE 3. On September 18, 1940, a 14-year-old Mexican boy was admitted to the San Diego General Hospital with the complaint of worms in his nose. The patient stated that on September 15, 1940, a fly flew into his left nostril while he lay half asleep in the fields. It was ten minutes before he could dislodge the fly from within his nose. On the morning of September 17 the patient noticed a bloody discharge from his left nostril, accompanied by frequent attacks of sneezing, both of which continued intermittently during the day, with the gradual development of a severe frontal headache. On the morning of September 18, the boy consulted a physician who removed 20 maggots from his left nasal cavity and sent him into the hospital.

On admission the physical examination revealed a well-nourished Mexican boy who complained of severe frontal pain. Anterior rhinoscopy revealed both nostrils containing much foul, purulent material. In the left nasal cavity several larvae were seen. The temperature was 100.8° F. and the white blood count was 11,500. A roentgenogram of the sinuses revealed a faint clouding of both antra and also a faint clouding of the ethmoidal areas. The patient gave a history of a foul smelling nasal discharge for several years.

On September 18, following the use of a chloroform spray, 50 larvae were removed from the left nostril with a bayonet forceps. These maggots varied in size from one-half to two-thirds inch in length. On September 19, the patient complained bitterly of pain in his left eye, and following use of chloroform spray ten larvae were removed. On September 20, the boy was still complaining of pain in his eye and six maggots were removed as well as several large pieces of necrotic bone and soft tissue. No more maggots were found and the patient was dismissed on September 30, after the temperature had been normal for two days. The patient received two grams of sul-

fathiazole on admission and one gram every four hours until his temperature had returned to normal.

CASE 4. The patient, a 56-year-old farmer, was admitted to the University of Kansas Hospitals on August 19, 1941, with the complaint of maggots in the right side of his nose. The patient dates the onset of his illness to August 15, 1941. While working in his hay field, a fly flew into his right naris and remained in his nose for five minutes. The patient began sneezing within 24 hours, and the sneezing was soon followed by a serosanguineous nasal discharge. Within the next 24 hours his right cheek became swollen and painful. He first blew maggots from his right nostril on August 18.

On admission the right cheek was swollen, reddened, and painful, and the right naris was literally crawling with larvae. The temperature was 103.4° F., and the white blood count was 15,250. The Wassermann and Kahn tests were negative. On August 19, 82 maggots were removed from the right naris with the use of a chloroform spray and bayonet forceps. On August 20, 35 larvae were removed in two attempts. On August 21, six maggots were removed and on August 22, one dead maggot was removed. On August 24, no more maggots were found, the swelling of the right cheek had disappeared, and the temperature had returned to normal. In addition, the patient received two grams of sulfathiazole on admission and one gram every four hours until dismissal, and supportive treatment. The patient gave a history of nasal catarrh for 15 years.

CASE 5. The patient was admitted to the University of Kansas Hospitals on September 19, 1941, with the complaints of bleeding from the nose, maggots in the nose, and swelling and redness of the left cheek. The patient dates the onset of his illness to September 16, 1941, when his eyes became inflamed and a serosanguineous nasal discharge began. On September 19, the patient blew a maggot from his nose. There was no history of a fly entering his nose or of his sleeping in the open. However, the patient, a farmer, drank heavily and was apparently unclean in his personal habits, so that it was possible that a fly could enter and leave his nose without the patient recalling it.

He had only mild discomfort. On admission the eyelids were slightly swollen, the conjunctivae were injected, and one live larva was found under the lower lid on the left. Anterior rhinoscopy revealed a large anterior perforation with maggots seen on both sides of the septum. This was the only case in which the larvae were seen in both nostrils.

There was a foul serosanguineous discharge on the left. The temperature was 102° F., and the white blood count was 13,500. The Wassermann and Kahn tests were negative. On September 19, 40 maggots were removed from the left nostril after the use of a chloroform spray. On September 20, 14 maggots were removed, and on September 21, six more maggots were removed. On September 22, five maggots were removed; the patient felt much better and the temperature was 99° F. On September 23, one dead maggot was removed and the patient was discharged on September 26, after two days of normal temperature. The patient was given two grams of sulfathiazole on admission and one gram every four hours until his temperature was normal.

SUMMARY

1. The screw-worm fly, Chrysomyia macellaria, is the usual etiological agent of nasal myiasis in this country, although other types of flies occasionally invade the nasal cavities of man.

2. Preexisting nasal disease, such as atrophic rhinitis and chronic purulent sinusitis, are definite predisposing factors in the development of this condition.

3. The diagnosis is often made by the patient and confirmed by the physician, who observes the larvae in the nose.

4. The chief symptoms and signs are: intense irritation in the nose with constant sneezing; a profuse, foul, serosanguineous nasal discharge from the affected side; pain, which is often very severe; moderate to high elevation of temperature; swelling of the affected side of the face; hemorrhage which occurs often and may be severe enough to require packing; tissue destruction within the nose which is usually extensive.

5. The treatment of this disease is simple and consists of chloroform applied to the nasal cavities by any one of several methods, combined with the mechanical removal of the larvae. Supportive and symptomatic treatment are helpful, as is the use of adequate doses of a sulfonamide, especially in those cases that have a high elevation of temperature.

6. Acute sinusitis, nasal hemorrhage, meningitis, and intestinal and bronchial myiasis are complications of nasal myiasis.

7. The prognosis of this disease is always serious, even fatal, if not recognized and treated promptly and correctly.

CONCLUSIONS

1. Nasal myiasis is not a common disease in this country, but it is not a rare one, especially in the Southwestern States. In the past twenty years, 21 cases of nasal myiasis have been reported in this country. Twelve of these were reported by R. J. Stroud of Arizona in 1927. Undoubtedly, there were many others that were not reported.

2. Acute sinusitis is such a common complication that it can practically be considered a part of the disease.

3. Sulfonamide administration appears to diminish the severity of the complications.

4. A clean healthy nose is apparently never invaded by the screw-worm fly.

5. All of the five patients in this series gave a history of a chronic nasal discharge accompanied by a foul odor for many years. One patient had a definite diagnosis of atrophic rhinitis made two years previously.

Three of these patients were seen occasionally in the clinic for several months after dismissal from the hospital. All three individuals stated that their nasal symptoms and signs were very much improved, and examination revealed very little nasal crusting and odor.

However, considering the seriousness of this disease this would, indeed, be a heroic treatment for atrophic rhinitis.

UNIVERSITY OF KANSAS HOSPITALS.

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XL

RHINOSCLEROMA: REPORT OF ONE CASE

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A comprehensive article on scleroma from the Mayo Clinic by Figi and Thompson[1] appeared in 1928 with a report of six cases which had been seen in the clinic. Since then cases have been reported by Canfield[2], Ireland[3], Chamberlin[4], Schwartz[5], Morrison[6], Simpson and Ellis[7], Gadomski[8], and Goldstein[9]. The last named author reports a careful search of the literature in the United States for the past fifty years and reveals 66 cases of rhinoscleroma, 8 of the patients being native born and 58 foreign born.

All authors agree that scleroma is a chronic disease of the upper air passage, which becomes manifest in the mucous membrane of the nose, nasopharynx or larynx. This disease may attack one or more of the above areas, and frequently the intervening areas of the upper air passages may be entirely uninvolved. Rhinoscleroma has three stages of development: (1) The edematous or catarrhal stage which resembles any chronic inflammatory reaction, and during which a diagnosis cannot definitely be made. (2) The granulomatous stage which produces an increase in the size of the lesion. During this second stage large vacuolated cells of Mikulicz, described also as foam or lace cells and containing Frisch's bacilli, are seen. Also frequent masses of hyaline tissue or Unna bodies enable the diagnosis to be made. (3) The final stage of fibrosis in which the granulomatous tissue is replaced by a firm contracting scar.

Treatment of scleroma has varied greatly, but most authors agree that roentgen or radium therapy in proper dosage offers the best hope of cure or control of this chronic infection. Peter[10] discusses treatment of scleroma with roentgen rays and advises that radiation be given very carefully, starting with a low dosage. The effect of radiation therapy is to produce the third stage of sclerosis and fibrosis.

REPORT OF A CASE

This case was seen in 1938 at the Henry Ford Hospital, the first case of rhinoscleroma in over 300,000 admissions.

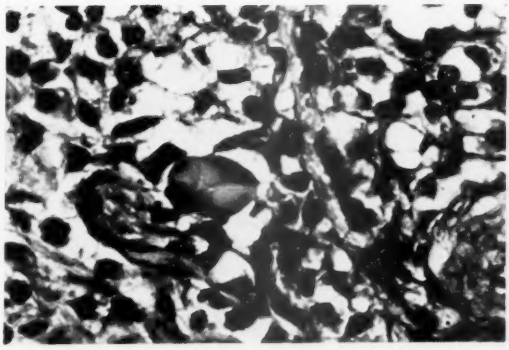

Fig. 1.—Photomicrograph, high power, of rhinoscleroma showing colloid cell in the center and many Mikulicz cells.

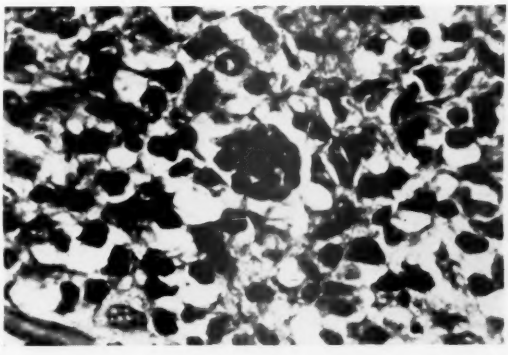

Fig. 2.—Photomicrograph, high power, of rhinoscleroma showing many Mikulicz cells.

Fig. 3.—Photograph taken in 1938 showing a granulomatous mass involving the upper lip, the columella and the vestibule of both nostrils.
Fig. 4.—Photograph taken in 1942 shows no evidence of tumor.

A Polish male of 48 years presented himself on January 17, 1938, for examination of a fairly painless tumor of the nasal septum and upper lip which he had first noted 18 months previously. The patient spoke English quite poorly so that a detailed history was difficult to obtain. The patient came to this country from Poland thirty years ago. He had always been exceptionally healthy.

The growth first appeared as an irregular mass in the right side of the septum and into the upper lip and began to occlude both nostrils. At no time had there been any marked pain or tenderness, but about a year previously the patient began to have frequent hemorrhages from the tumor. Lately it had begun to ulcerate to a slight extent.

The general physical examination was negative; blood hemoglobin was 100 per cent; red blood count 5,000,000; white blood count 8,800; polymorphonuclears 86 per cent. The urine and blood Wassermann tests were negative.

Locally (Fig. 3) there is a firm indurated mass apparently originally springing from the junction of the columella and the upper lip. It has almost occluded both nares and extends quite far down on the

upper lip. The surface is quite soft and crusted over in spots as though slight ulceration has taken place. Both alae are extremely rigid but show no signs of the tumor externally. The remainder of the nose and throat examination was essentially negative. Nothing unusual was noted in the pharynx or the larynx.

The clinical diagnosis (pre-operative) was: chronic granuloma, etiology unknown.

Biopsy of a specimen taken under local anesthetic was reported as rhinoscleroma.

Treatment consisted of deep x-ray therapy.

Fibrosis has taken place with marked stenosis of the left nostril. There is considerable fibrosis throughout the left nostril with crusting. The soft palate is fibrosed and the uvula is turned upward and adheres to the superior surface of the soft palate.

The patient has remained well these past four years with no evidence of recurrence of the growth. (Fig. 4).

The Pathological report (microscopic) is as follows: Section through the tissue received shows a portion of the skin and the underlying connective tissue including a few striated muscle fibers. The surface of the skin in many areas shows almost complete ulceration, only a few small groups of squamous cells remaining. The main portion of the tumor consists of large masses of plasma cells whose morphology varies moderately in the different sections examined. Many of the plasma cells show varying degrees of degeneration. Most of them have very typical cartwheel nuclei and light pink or pinkish-blue cytoplasm. Larger reticulo-endothelial cells are also visible throughout the structure. In addition there are very large oval cells which have a foamy lacy cytoplasm and a small pyknotic nucleus displaced to one side. These are the so-called Mikulicz's cells typical of this lesion. In addition, large oval cells, having a dense pink cytoplasm and a pyknotic degenerated nucleus, are noted. These are the so-called colloid cells typical of this lesion. In addition numerous Russell bodies or degenerated plasma cells without morphology are noted. The fibrous stroma of the tumor is small in amount, and a moderate number of blood vessels, mainly capillaries, are noted here and there. Portions of the tumor appear to be infiltrated by polymorphonuclear leucocytes and suggest beginning abscess formation in a few areas. The tumor is infiltrating the muscle fibers in some areas.

HENRY FORD HOSPITAL.

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XLI

CHRONIC GRANULOMATOUS ULCER OF THE NOSE OF UNKNOWN CAUSE

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This discussion will relate largely to the report of a case of chronic granulomatous ulcer of the nose of which the cause is not known. It is believed that the granulomata of specific cause have been reasonably well excluded and that this case falls into that group of four cases of granulomatous ulcer of the nose and face discussed by Hoover[1] two years ago. These cases were progressive, gangrenous, and fatal and were of unknown cause. In the fifth case, which he described as atypical the patient apparently recovered.

Previously, according to Hoover's survey, only two cases had been reported in American literature, although approximately thirty cases had been reported abroad. The first case reported in America was by Wood,[2] in 1931. In this report he presented a case of "mutilating granuloma of the nose and face with a fatal ending." The second was by Lewy,[3] in 1938, who reported "a case of gangrenous osteomyelitis of the paranasal sinuses." In 1942, Lierle[4] reported a case of "chronic infectious granuloma of the nose," of unknown cause.

Brief mention may be made of several conditions more or less common to the cases reported:

A much greater incidence in males, and in middle age, although young and old are affected.

Insidious onset and often a course of several years.

Tendency to periods of quiescence, and even apparent recovery.

Destruction of the soft tissues and the subjacent bone and cartilage.

A peculiar mucopurulent, sometimes gelatinous, exudate, with marked crusting and extremely foul odor.

Read before the Southern Section Meeting of the American Laryngological, Rhinological, and Otological Society, Chattanooga, Tenn., January 28, 1943.

Frequent involvement of the tissues in relation to the lacrymal sac.

Irregular excursions of temperature between normal and 105° F.

Absence of depression and exhaustion to be expected in the presence of such a lesion.

Absence of any characteristic blood picture, bacteriological or other laboratory findings.

Diagnosis necessarily by exclusion and the clinical course.

The diseases necessary to exclude are: syphilis, tuberculosis, malignant diseases, leprosy, anthrax, the typhoid group, myiasis, noma, fungus infections, agranulocytosis, glanders, diabetic gangrene, tularemia.

REPORT OF CASE

On Oct. 21, 1940, the patient, a white man, aged 62, was first seen in my office. He complained of feeling "thick headed," and of constant soreness in the throat for about three weeks. He also complained of his nose being stopped up and of postnasal discharge.

During the summer of 1940 he had been treated by another physician for a "severe sinus infection." The sinus was treated, at least in part, by irrigation. Ten years previously the patient's "sinuses" had been operated on by still another physician, and he had been entirely free of symptoms for five years. During the past five years he had nasal obstruction and discharge at gradually increasing intervals.

Examination showed all his teeth to be absent. The tonsils were fairly prominent but not injected. The uvula was very large, thick and somewhat red. Mucopurulent discharge hung on the posterior nasopharyngeal wall. The nose was rather wide open on both sides, with widely distributed foul crusts and pus underneath them. There was a definite amount of atrophy of the soft tissues. Both frontal sinuses were large and transilluminated equally and well. The right antrum was completely dark. The left antrum transilluminated fairly well. Irrigation fluid returned from the right antrum was clear. *Impression*: Subacute upper respiratory infection in a patient with atrophic rhinitis.

Home treatment of saline irrigations and a spray of glucose in glycerin was instituted.

Four days later the temperature was 99° F. The patient felt better, although he reported that his temperature had reached 103° F. The throat seemed infected. Sulfanilamide was prescribed and continued for several days with lowering of the temperature and apparent improvement.

On November 7, 1940, an ulcerated area beneath a large foul crust was first discovered on the posterior aspect of the right side of the septum. The tissues about this ulcer had become thickened, especially about the upper septum and the posterior nares. The border of the ulcer possessed a peculiarly gelatinous, semi-necrotic appearance and the odor was foul. Biopsy specimens from the border and other thickened areas showed "an infiltration of small round cells and some areas in which the surface epithelium was denuded. "There is no evidence of malignancy or tuberculosis. Microscopic diagnosis: Chronic inflammatory changes."

Serological tests for syphilis were negative. Repeated cultures and smears showed at various times staphylococci alone and at other times various organisms including staphylococci, streptococci, and diplococci. Usually the culture showed a nonhemolytic staphylococcus albus alone. No tubercle bacilli could be found. Smears and cultures on Sabouraud's agar were negative for fungi.

Agglutinations for the typhoid group, tularemia, and undulant fever were negative. The tuberculin test was faintly positive. The blood examination at this time showed 5,100,000 red cells, 7,050 white cells, hemoglobin 82 per cent, color index .8, and a normal differential.

The weight was 220 pounds. The blood pressure was 170/100. X-ray examination of the chest showed the heart enlarged to the left. No evidence of tuberculosis was found. The urine examination showed one plus albuminuria, occasional cast, no sugar, and no blood. Sinus x-ray films showed moderate clouding of both antra, and slight clouding of the anterior ethmoids. The posterior ethmoids and sphenoids were clear.

The patient felt well despite several rises of temperature to as high as 103° F.

Various treatments were used locally, at different times, including a solution of bismuth violet, merthiolate ointment, sulfathiazole ointment, sulfanilamide powder in conjunction with mechanical cleansing and saline irrigations. X-ray therapy was given. Sulfathiazole was given by mouth. At other stages intravenous in-

Fig. 1.—Showing perforation of hard palate.

jections of neoarsphenamine and rather large doses of potassium iodide were given.

No treatment seemed to have any marked or specific effect, either locally or on the patient's general condition. The temperature rises were not affected. Sulfonamides were at all times tolerated well.

On February 1, 1941, a sequestrum 1 cm. long and nearly as wide was removed from the area of the ulcer. A month and a half later the lesion was apparently healed. There was only a slight tendency to crusting and little or no indurated tissue. The patient was dismissed.

Nine months later, Dec. 1, 1941, the patient, who had remained well until a few days before, was seen with what appeared to be an acute dacryocystitis on the left. There was marked brawny swelling and redness about the left side of the root of the nose and about the area of the lacrymal sac, with marked edema of the lids. There was very little soreness or pain. Irrigation through the lacrymal system was readily accomplished, although there was slight tearing. Induration of the tissues with the same characteristic crusting, necrosis and foul odor became rapidly evident on the left lateral wall of the nose. The temperature ranged from normal to 103° F. but the patient felt surprisingly well.

After three weeks an external incision was made but no pus was obtained. A purulent discharge did appear within three days and the wound was kept open with a small pack. Treatment similar to that used during the original condition was instituted until January 22, 1942, when a portion of the frontal process of the left

maxilla, which had become bared, was removed. A rather large opening into the nose was made and the wound closed externally. A month later the external tissues were nearly normal and there was much less crusting and induration of the tissues within the nose. The patient was told to continue treatment at home and to return in three weeks.

The patient was seen three months later with no complaint. There was moderate atrophy in the nose and a minimum of crusting, but no grossly active lesion.

On Sept. 15, 1942, (8 months after the operation) the patient returned with the same type of foul crusty condition on both sides of the cartilaginous septum, more marked on the left where there was a superficial ulceration. The lesion gradually destroyed most of the septum, and spread across the floor of the nose on both sides and to the lateral wall on the right. At this stage, early November, 1942, marked brawny swelling and redness occurred over the surface of the right antrum, adjacent to the nose, and across the upper lip. Swelling and redness of the hard palate and gum margins were present and there were necrosis and perforation of the hard palate. The temperature rose above 105° F. and the patient for the first time during the long course evidenced a marked degree of exhaustion. He had lost 40 pounds and the odor was extreme. His death seemed probable, and not far off.

No treatment had any beneficial effect. He was again given x-ray therapy in conjunction with the use of a thick caroid (carica papaya) paste and potassium permanganate irrigations. Most of the crusts, foul odor, and discharge disappeared rapidly. The borders of the lesion were much less active in mid-January, 1943. However, the floor of the nose on both sides and part of the lateral wall of the nose on the right presented areas of bare bone and an enlarging perforation of the palate.

At this time the patient's temperature has returned to normal. He is stronger and has regained 20 pounds. Red and white blood counts are normal but there are 82 per cent polymorphonuclears, all segmented.

Although this routine served best of all in cleansing the lesion, there is no probability of any other effect. It seems more likely that this is another period of quiescence. Absence of the trauma incident to mechanical cleansing may be an advantage of this routine, inasmuch as the slightest trauma seems to favor spread. Urea as a proteolytic substitute for caroid was not tried.

During the course of observation repeated biopsies, serological tests for syphilis, examinations for tubercle bacilli, B. mallei, and fungi have been made without yielding positive information as to the cause.

In January, 1943, a guinea pig was inoculated by an injection into the subcutaneous tissues of the right flank and the perineal cavity. Approximately three months later, an autopsy was performed on it.

The pathological report was as follows: A section of liver showed some congestion and cloudy swelling. A section of the abdominal wall showed a large area of necrosis and marked round cell infiltration and fibroblasts. No tubercle formation was found. A section of the lung showed congestion, perivascular round cell infiltration, thickening of the alveolar walls and pleura and collections of lymphocytic cells but no evidence of tubercle formation. Smears from the wall of the abscess and the abdominal wall showed no tubercle bacilli.

SUMMARY

An additional case of chronic granulomatous ulcer of the nose is presented, in the hope that we may be more conscious of its possible occurrence and mode of action.

The complete lack of any favorable effect from sulfonamides used locally and generally suggest that the usual pyogenic organisms are not an etiological factor.

In this case, the use of proteolytic agents, potassium permanganate irrigations, and the avoidance of trauma, through the use of x-ray, have offered the only worthwhile treatment in the absence of a specific therapy.

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XLII

THREE UNUSUAL CASES OF HEMANGIOMA

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The consensus of opinion is that most hemangiomas in children are best treated by irradiation. Figi[1] recommends surface irradiation, interstitial radium element needles or interstitial radon, depending on the nature of the lesion. Brown and Byars[2] suggest the use of radon seeds valued at 0.25 millicurie for interstitial irradiation. Dr. H. P. Doub and the writer have recently successfully treated a case using eight gold radon seeds of that value in a cavernous hemangioma about 3 cm. in diameter. The tumor involved the lobule of the ear and the posterior auricular region of a child eleven months old. This tumor had previously had roentgen treatment by Dr. Doub with definite improvement, especially in the superficial capillary portion.

Edwards[3] reports good results from the surface application of beta radium rays in certain cases and the use of interstitial low radium content monel metal needles in others. Salinger[4] states, "Surface irradiation, while successful in the treatment of superficial angiomas of small size, could have little effect on a deep-seated vascular growth unless applied in such doses as to endanger the integrity of the overlying tissues." The case he reported was in an adult, however.

Spencer[5] feels that the use of high voltage roentgen rays is equally effective and more convenient than radiation. Hodges, Snead and Berger[6] feel that contact roentgen therapy is the method of choice in treating tumors of this type.

Edwards[3] feels that dry ice is valuable for treating small hemangiomas and may be useful around the periphery of certain of those previously irradiated. Wrong[7] recommends carbon dioxide snow for small, thin hemangiomas. Poppe[8] reports a method of treating certain of these tumors by a cold air blast which is produced by blowing a current of compressed air through a coil of metal tubing packed in carbon dioxide ice.

Since the radio resistance of these tumors increases with the age of the patient, the treatment of them in adults presents a different

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Fig. 1.—Pulsating cavernous hemangioma.

problem. Certain of the small superficial lesions can be excised. Those which cover a larger area may require excision in stages, while others must be covered by a free or pedicled skin graft.

Figi[1] says, "From time to time, especially in adults, one encounters a localized angioma that can be removed most satisfactorily surgically, but cases of such a growth are decidedly in the minority." In the past two years the writer has seen two hemangiomas attached to the nasal septum and one attached to the anterior end of the inferior turbinate. These were all successfully excised.

Figi says, "Electrocoagulation is the treatment of choice in many instances of cavernous hemangioma in an adult." He uses an electrode which is insulated except at its distal end. In his opinion this method has largely supplanted the use of radium in treating certain of these tumors in adults.

The injection of sclerosing solutions is the method of choice in treating certain of these tumors. Watson and McCarthy[9] use a five per cent solution of sodium morrhuate when injection treatment is used. Bellinger[10] recently successfully treated a large cavernous hemangioma of the tongue in an adult with this method.

The following cases are reported because they are rarely encountered. A study of the literature for the past ten years reveals relatively few case reports of similar conditions. In a report of 1308 hemangiomas in 1001 patients, by Watson and McCarthy,[9] I could

find no mention of similar pathological entities. In each case there was a potential or an actual threat to the general health or the life of the patient. Surgical removal was successfully carried out in all cases.

PULSATING CAVERNOUS HEMANGIOMA OF THE EXTERNAL EAR

Faleyeva[11] reported a case of pulsating angioma racemosum involving the external ear of a ten-year-old boy. The condition had first been noticed at the age of two. Since the tumor had hemorrhaged, the external carotid artery was ligated. This was followed by diminution in the size of the tumor.

It is felt that in the following case, aside from the unpleasant appearance, the danger of troublesome hemorrhage from ulceration or trauma was imminent.

CASE 1.—A white man, aged 19, presented himself at the Henry Ford Hospital on June 10, 1941. He stated that there was a tumor involving his left external ear which had been present since birth. Three or four months previously the tumor had appeared to increase in size and it had begun to throb. There was no history of other ear, nose or throat disease of any consequence. He was interested in having this tumor treated both from the standpoint of appearance and also because he had worried some about the increase in size and the throbbing.

On examination of the left external ear, there was noted a large pulsating tumor which was bluish in color, was compressible, and appeared to be covered by rather thin skin. It appeared that the skin itself might be involved by the tumor. The involved area included practically all of the anterior surface of the external ear, except the lobule. Pressure over the posterior auricular and the superficial temporal arteries resulted in cessation of the pulsation of the tumor.

The remainder of the ear, nose and throat examination, the general physical examination, and the laboratory studies revealed no significant abnormalities.

A diagnosis of pulsating cavernous hemangioma of the left external ear was made. (Fig. 1)

On June 13, 1941, under local anesthesia, consisting of an injection of one per cent novocaine, the auricular branch of the superficial temporal artery and the posterior auricular artery with their corres-

Fig. 2.—Roentgenogram showing cloudiness of the right mastoid.

ponding veins were all ligated. After ligation of the auricular branch of the superficial temporal artery most of the pulsation in the tumor stopped, although there was still slight pulsation synchronous with the heart beat. It was felt that the tumor could either be removed or injected with one of the sclerosing materials, probably sodium morrhuate, and the choice of treatment depended upon the condition of the skin overlying the tumor. It was felt that if the skin were sufficiently healthy, the tumor could be removed and the skin used to cover the anterior surface of the ear; whereas, if the skin were too much involved by the tumor, injections would probably be better.

The skin was found to be of sufficient thickness and good enough quality to cover the anterior surface of the ear, so a long incision was made through the skin close to the helix and practically along its entire length. Then the skin was dissected backwards toward the helix and forward toward the external auditory canal. The tumor was removed completely, cleaning all remnants of it thoroughly from the perichondrium and also from the overlying skin. The skin was then closed with fine silk and a pressure dressing applied. One small rubber drain was inserted.

The postoperative course was uneventful except for slight oozing of blood from the incision when the dressing was changed. The ear was rather thick at first, but it gradually shrank to approximately the size of the other ear. On July 1, 1941, the dressing was left off and the patient was dismissed.

CAVERNOUS HEMANGIOMA OF THE MIDDLE EAR AND MASTOID

There are relatively few cases of hemangioma of the middle ear and mastoid reported in the literature. Hampton and Sampson[12] reported two cases of hemangioma of the tympanic cavity. The diagnosis was made by biopsy, and roentgen treatment was carried out. McKenzie[13] reported a case of hemangioma of the middle ear and petrous bone. A biopsy was not made, however.

Since in the following case the tumor had produced signs of labyrinth irritation, it was thought that it constituted a definite threat to the health of the patient. In addition, pain was a prominent symptom.

The exact nature of the tumor was not known until a microscopic examination of frozen sections was made.

CASE 2. A 56-year-old white man was examined July 8, 1942. He stated that ten years previously he had developed a severe pain in the right ear. This had lasted about a week, during which time it had been discovered that there was an obstruction in the right external auditory canal and that the hearing in the right ear was markedly impaired. The pain had recurred at frequent intervals thereafter until two years before examination, at which time he had had an attack of dizziness which was sufficiently severe to cause him to stagger from side to side while walking. There had been anorexia but no nausea or vomiting. This had lasted for about four weeks. There had been no recurrence of the dizziness, but the pain, which was in the region of the right mastoid and occiput, had become more persistent and was sufficiently severe to disturb his sleep at times.

On examination a tumor was seen completely obstructing the right external auditory canal. It was attached to the anterior bony canal wall and was covered by the skin of the membranous canal wall. It was moderately compressible in its posterior half, but the anterior half felt bony and firm through the skin. There was no tenderness to pressure over the right mastoid process but there was tenderness to percussion in this area. Pain in the right temporomandibular region could be produced by certain movements of the lower jaw. There was marked combined deafness in the right ear. The hearing was normal in the left ear. The ear, nose and throat examination revealed no other significant abnormal findings.

Roentgenograms showed the left mastoid to be normal but there was evidence of considerable cloudiness and destruction of the cellu-

Fig. 3.—Roentgenogram showing cloudiness of the right ethmoid and maxillary sinuses.

lar outline of the right mastoid. There was also obliteration of the right external auditory canal shadow (Fig. 2). The neurological examination was objectively negative. The general physical examination and the examination of the blood and urine revealed no significant abnormalities.

On July 10, 1942, under ether and nitrous anesthesia, the cortex was removed from the right mastoid. There was moderate bony sclerosis present. When the antrum was exposed a tumor was seen which filled it. The middle ear was also practically filled by the tumor and it was found to be rather securely attached to the anterior bony auditory canal wall. Slight manipulation of the tumor released a small amount of clear cystic fluid. It was then removed in its entirety.

Since the nature of the tumor was not known, the wound was not closed until microscopic examination of frozen sections had re-

vealed that it was an hemangioma. The tumor itself had produced practically a radical cavity, so without much further surgery a radical skin flap was made, the cavity packed with vaseline gauze, and the wound closed.

The specimen measured 18 x 11 x 10 mm., but it was somewhat larger than this before the release of the cystic fluid.

Microscopic examination of fixed sections of the hemangioma revealed a thin squamous epithelial surface having a narrow keratotic layer. There were none of the usual skin structures present and no papillary formation. A few bony spicules were present which were apparently in the region of its attachment to the anterior auditory canal wall.

The patient was dismissed from the hospital on the eighth post-operative day. When last seen, seven months after the operation, the radical mastoid cavity was well epithelialized and dry. He had been completely free of symptoms.

CAPILLARY HEMANGIOMA OF THE ETHMOID SINUS REGION

Hemangiomas occurring in the nasal cavity and the paranasal sinuses are sufficiently uncommon to constitute a diagnostic problem when they do occur. For this reason it is felt that the following case is worthy of a report in this discussion.

Irradiation was used as an auxiliary measure.

CASE 3.—A white woman, aged 38, was admitted to Henry Ford Hospital on September 24, 1940. There was a history of recurring hemorrhage from the right nasal cavity during the preceding twelve months. A month previously examination elsewhere had disclosed a hemoglobin of 37 per cent and a red blood cell count of 3,100,000. Two blood transfusions of 500 cc. each had been given at an interval of several days and a section for biopsy had been taken from the right antrum through a Caldwell-Luc incision. This had been reported as chronic sinusitis with edema.

The hemorrhage had recurred and during the few days previous to our first contact with her the bleeding had been almost continuous. The blood examination at the time of our first examination showed hemoglobin 9.5 grams; red blood cell count 3,400,000; white blood cell count 4,950; bleeding time 3 minutes; clotting time 5 minutes 20 seconds; fragility: initial hemolysis 0.44%, complete hemolysis

0.32%; vitamin C 1.12 mg. per 100 cc.; prothrombin 100%; and platelets 315,000.

The general physical examination and the remainder of the laboratory studies revealed no significant abnormalities.

The ear, nose and throat examination showed a bleeding point in the region of the right ethmoid bulla. Since the examination was otherwise negative, a section for biopsy was taken from this region. This was reported as capillary hemangioma. Roentgenograms of the sinuses showed diffuse cloudiness of the right maxillary and ethmoid sinuses (Fig. 3).

Following the removal of tissue for biopsy, several weeks elapsed before the hemorrhage recurred.

On October 18, 1940, with local and nerve block anesthesia, a right ethmoidectomy was carried out. The cells were found to be rather badly infected. A diseased portion of the right middle turbinate was removed. The right antrum was then opened through a Caldwell-Luc incision and the thickened, badly diseased lining membrane of the antrum removed. A large nasoantral window was constructed. A 50 mg. tube of radium in a filter was placed in the right ethmoid area and left in for four hours. It was felt that this dose would produce some scarring in this area.

Further evidence of the hemangioma was not found microscopically in the infected tissue which had been removed.

The postoperative course was uneventful. Three months later, when last seen, there had been no recurrence of the hemorrhage and there was no evidence of infection in the nose or the sinuses.

COMMENT

The best results are obtained in the treatment of hemangiomas by a consideration of all the accepted methods of treatment and the application of the method or combination of methods which is most efficacious for the particular characteristics of each case. Factors such as availability of material and the experience of the operator must also be considered.

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XLIII

DENTIGEROUS CYST OF THE MAXILLARY ANTRUM

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At the suggestion of H. I. Lillie, I[1] once reported the case history of a patient from whom he had removed a dentigerous cyst of the maxillary antrum which contained an unerupted third molar tooth. When first seen this patient had a cellulitis of the cheek which had followed an attempt at antrum lavage by his local surgeon. The cyst was removed about three weeks after the acute infection had subsided.

There were two reasons for reporting that case: first, a dentigerous cyst associated with an unerupted third molar tooth is rare, and second, the case illustrated the fact that lavage of the antrum without first having roentgenograms of the sinuses involves some risk.

Millhon and Williams,[2] commenting on a somewhat similar condition in a man 48 years old, state: "A dentigerous cyst associated with an upper third molar tooth in persons of this age is one of the rarest pathologic conditions that occur in the jaws."

The following case is thought to be of interest, first, because of the apparent infrequency with which this condition is encountered; and second, because, since the diagnosis was made by roentgenograms before any treatment was carried out, the chance of acute infection from antrum lavage was not taken. This is further evidence that unless the presence of some unusual condition in the antrum can be completely excluded by physical examination, it is best to have roentgenograms made of the sinuses before carrying out antrum lavage.

A white man, aged 35, was examined at the Henry Ford Hospital on October 8, 1942. He complained of frontal and occipital headache which had been present intermittently for about a year. Its occurrence had been more frequent during the preceding few weeks. It was not relieved by the usual dose of aspirin and was severe enough to awaken him during the early morning hours on a number of occasions. There had been slight transient dizziness. An ophthalmological examination elsewhere had shown no abnormality. There was no history of tooth extraction.

Fig. 1.—Roentgenogram of sinuses showing cyst wall and unerupted third molar tooth; posteroanterior view.

Fig. 2.—Roentgenogram of sinuses; lateral view.

Fig. 3.—Cyst wall and tooth.

Examination of the ears, nose and throat revealed that the tonsils had been removed and that the left antrum was cloudy on transillumination. Roentgenograms of the sinuses showed a cystic tumor in the left antrum which appeared to contain an unerupted tooth (Figs. 1 and 2). Dental roentgenograms showed the absence of the left upper third molar tooth.

On general physical examination the blood pressure was found to be systolic 140, diastolic 90. A right inguinal hernia was present. The right testicle was undescended. The examination was otherwise negative. The hemoglobin was 100%, the leukocytes 8,250. The blood Wassermann test was negative, and the urinalysis showed no abnormalities. The basal metabolic rate was —8%.

A diagnosis was made of dentigerous cyst of the left antrum, containing an unerupted third molar tooth.

On November 27, 1942, with local and nerve block anesthesia the left antrum was opened through the Caldwell-Luc approach. A cyst about the size of a black walnut was readily seen. This was

covered almost completely by a thin eggshell-like layer of bone. The thick milky material was evacuated and the cyst wall removed. A well-attached tooth from which the cyst had developed was removed from the wall of the antrum (Fig. 3). A large naso-antral window was made.

On microscopic examination the cyst wall was found to consist of fibrous tissue lined by squamous epithelial cells which were flattened and attenuated. Considerable inflammatory infiltration was present. The pathological diagnosis was dentigerous cyst.

The postoperative course was uneventful and the patient was dismissed from the hospital on the third postoperative day.

On examination six weeks later the condition of the nose appeared normal and there had been no recurrence of the headache or dizziness.

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XLIV

CONGENITAL FAMILIAL DEAF-MUTISM IN SIX CHILDREN

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Heredity is a fifty-fifty proposition in which both partners may be responsible for the future disposition (anlage) of the child. If the determining units or genes, according to Mendel's law, appear equal they are called homozygotic. This means that the father and mother are equally healthy or ill. If these units differ, that is, if the father is ill and the mother is healthy, or vice versa, we speak about heterozygotes.

If the ill qualities of the child are distinguishable or manifest we call them dominant; if they are latent or not distinguishable we call them recessive. In the development of sexual cells the paired anlage is divided (separated) and a heterozygotic person would carry over the healthy factor to one half of his children and the ill factor to the other half. In our case both parents have normal hearing and are in good health. One of them may be a latent or recessive carrier of deafness.

REPORTS OF CASES

(The B. Family)

There is a negative history of deafness on the side of both the mother and the father.

Mr. B. This man, aged 37, brown-eyed, hears well. He has had measles, whooping cough and bronchitis. At present he has heart trouble, possibly angina pectoris. His gall bladder was removed July 1942. The serological test was negative. His hearing is normal for voice and tuning forks. The vestibular test and the audiogram are normal.

Mrs. B.: This woman, aged 34, has nine children. She was in good health until her last baby was born in May 1942. At that time she developed high blood pressure and kidney trouble. There is no history of any childhood diseases. The serological test was negative.

Read before the Chicago Laryngological and Otological Society, March 1, 1943.

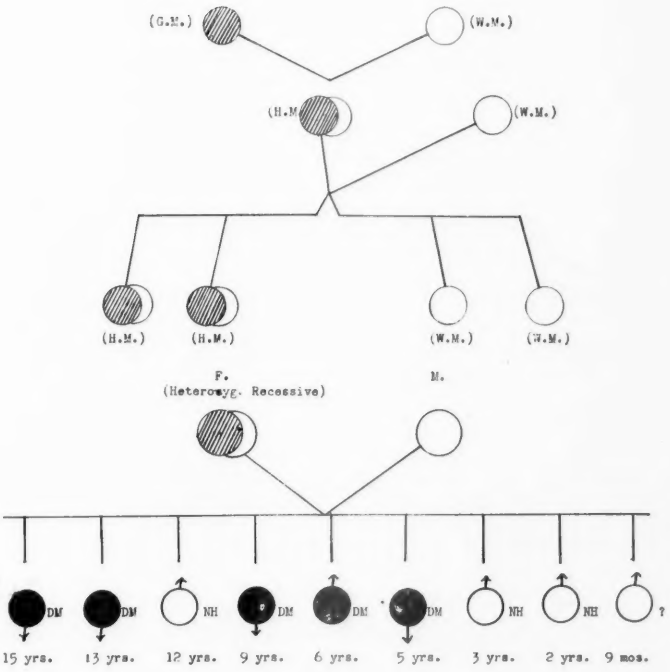

Fig. 1—G.M., grey mouse; W.M., white mouse; H.M., heterozygotic mouse; DM, deaf mute; NH, normal hearing; F, father; M, mother.

She hears well. The tuning fork test, the voice test and the audiogram are normal. The vestibular test is normal.

Genevieve: This 15-year-old blue-eyed girl had scarlet fever at the age of three, followed by a right mastoid operation; mumps at the age of six; measles at seven; no other illness. This child did not speak at all until the age of seven, and now her speech is defective. Only lately has she shown any signs of hearing. She enjoys listening to the radio.

Her ear drums are retracted and atrophic. Weber's test lateralizes to the right; Rinne's test on both sides is infinitive, negative; Schwabach's test is diminished. She hears a loud spoken voice to the ear, and speaks only in monosyllables.

Geraldine: This 13-year-old, blue-eyed girl had measles and whooping cough at the age of four. She did not speak until the age of six but now speaks fairly well. She has considerable stomach trouble.

Examination of the right ear showed an anterior pin-point drum perforation but the ear is dry. The ear drum is retracted. The tonsils are large; the nasal mucosa pale. Weber's test does not lateralize, and Rinne's test is positive. Bone conduction is diminished. The low and high tones are highly reduced. The vestibular test is normal. She speaks in monosyllables.

Robert: This 12-year-old, blue-eyed boy had whooping cough and measles at the age of three; chickenpox at the age of eight. There is no history of any ear trouble. He spoke at the age of two and a half, was always alert, and heard well.

Examination revealed normal drums, the nose negative, and the tonsils enlarged. The tuning fork test was normal. He hears a whispered voice at twenty feet bilaterally. The responses to turning vestibular tests are normal, the audiogram shows normal hearing.

Marie: The only illness of this 9-year-old, brown-eyed girl was German measles. She did not speak at all until six or seven, and had no previous ear trouble.

Examination showed both drums retracted, a slight crusting of the nose, and large tonsils. She speaks in monosyllables. Weber's test does not lateralize, and Rinne's test is positive. Bone conduction is present only with a highly reduced spoken voice ad concham. Responses to turning vestibular tests are normal.

Donald: This 6-year-old, brown-eyed boy, who is just starting to speak, has always been in good health.

Examination revealed retracted drums. Bone conduction is present with the spoken voice ad concham. The fork tests are difficult to explain. The labyrinthine test after turning is normal. This child speaks only in monosyllables.

Alice: This 5-year-old, brown-eyed girl has had no previous illnesses. She does not speak at all and has poor hearing.

Examination showed retracted drums. She tries to talk but produces only monosyllabic words. Fork examination is not accurate. Turning vestibular test is normal and there is no dizziness afterwards.

James: This 3-year-old, brown-eyed boy has had no illness. He spoke at the age of two.

Examination revealed normal drums. The fork examination was unsatisfactory; the turning test was normal. The audiometric examination was unsatisfactory because of the child's inability to cooperate. He speaks fairly well for his age.

Frank: This 2-year-old, brown-eyed boy has had no illnesses.

Examination revealed normal drums. He speaks satisfactorily for his age.

Thomas: This child is 9 months old. The mother states she believes the child hears her.

In similar cases of deaf-mutism the anatomical configuration of the bony cochlear capsule was found to be normal. The pathological changes were situated mainly in the stria vascularis, in Corti's organ and in the ganglion of Scarpa. However, the most important findings were pathological changes in the nuclei of the auditory nerve and partly in the central pathways.

The form of communication was not exactly studied but we found that the older children are lip readers. They talk with their siblings who hear well by producing words, often pronouncing them correctly. With the younger deaf children communication takes place by means of pointing and gestures. Generally speaking, their language symbols are poorly developed. In fact, they recently started to attend special school.

All the children have shown normal vestibular reaction, considering the fact that the labyrinth plays an important part in the articulation. We also have found that deaf-mutes with a normal acting labyrinth are, first, more fit for early lip reading and second, learn to talk more readily, making fewer errors in articulation. They also acquire a sense of rhythm which consequently gives them a fairly intelligible speech; lack of monotony in verbal expressions and a right accentuation can even be achieved.

30 North Michigan Avenue.

XLV

ORBITAL CELLULITIS COMPLICATING A MAXILLARY SINUSITIS

EDWIN J. BLONDER, M.D.

CHICAGO, ILL.

Orbital cellulitis complicating a sinus infection is not uncommon, but orbital cellulitis following an antrum infection is relatively infrequent, according to a review of the cases which have been reported in the literature. The records show that orbital involvement in children results from ethmoid infection more frequently than from infection in the other sinuses, while in adults infection in the frontal sinus is the most frequent cause of orbital involvement. The extension of the infection from the sinuses occurs through venous channels, through dehiscences or osseous necrotic changes in the orbital wall.

CASE 1.—The patient I am reporting is a boy, aged 14 years, who entered my service at the Illinois Eye and Ear Infirmary on January 14, 1943, with proptosis of the right eye and swelling of the periorbital tissues, more pronounced at the lower lid. The history obtained was that the boy had a head cold for about two months and a swollen right orbit for about five days before entry to the hospital. No intranasal treatment had been given. In addition to the proptosis, the conjunctiva was edematous and moderately injected. No limitation of motion of the eyeball was present. The right naris was filled with thick yellow pus. The face over the antrum appeared full and was tender to palpation. The temperature on admission was 100.2° F. The white blood count was 23,600, of which 82% were polymorphonuclear leucocytes, 15% lymphocytes, and 3% monocytes. The red cells amounted to 3,840,000 with 64% hemoglobin. The blood Wassermann was negative. The Mantoux reaction was negative. Ophthalmological consultation reported no intraocular changes. The roentgenological report was as follows: "The right antrum is definitely clouded with reactionary osteitis in the floor and medial wall. The nature of the opacity suggests an inflammatory change, such as mucosal thickening, and the presence of fluid. The right ethmoid is definitely clouded, and extends posteriorly and superiorly to the orbit."

Read before the Chicago Laryngological and Otological Society, March 1, 1943.

Our diagnosis was orbital cellulitis resulting from a sinus infection. Early thrombosis of the cavernous sinus was considered.

Our original management was conservative. The boy was put to bed. Hot applications were applied to the affected side. Fifteen grains of sulfathiazole were given by mouth every four hours. Drops of one per cent ephedrine solution were prescribed for the nares; cocaine-ephedrine packs were placed in the middle meatus, followed by suction several times daily. After five days of this conservative management, the proptosis and swelling of the right eye increased, and limitation of movement of the eyeball upwards developed. The white count remained high, 22,200, and the boy appeared increasingly ill. It was apparent that the conservative management was not limiting the periorbital infection and that more radical measures must be instituted.

The question of orbital abscess or cellulitis could be answered only at operation. Thrombosis of the cavernous sinus could not be definitely ruled out, but seemed less likely because of the absence of intraocular findings. The second question involved the method of procedure, namely, whether the antrum or the ethmoid was responsible for the complication, since both the antrum and the ethmoid were involved. There was practically no swelling or tenderness at the inner canthus, and the eye was not directed downwards or outwards. Most of the swelling occurred at the lower lid, and in addition, limitation of the eye movement upwards was present. Considerable tenderness and induration over the antrum was elicited. Therefore, I felt that it was reasonable and likely that the right antrum was the origin of the severe orbital complication.

On January 19, 1943, five days after the patient's entrance to the hospital, under general anesthesia, a gingivolabial incision was made on the right side, the periosteum elevated, and the anterior wall of the antrum was widely removed. The antrum contained about a teaspoonful of fairly thick yellow pus. The mucous membrane throughout was markedly injected and necrotic. There was a considerable amount of angry granulations at the orbital surface of the antrum, but no perforation could be probed. The pus, granulations, and necrotic tissue were removed. The ethmoid cells were exenterated through the antrum to lessen the possibility of reinfection of the already devitalized antrum. No orbital incision was made, and the orbital surface was not removed. An antrum window was then made under the inferior turbinate. Powdered sulfanilamide was placed in the antrum. No pack was inserted. The wound was approximated, but not sutured. A section of the anterior wall, a portion of the

mucous membrane, and a specimen of the pus were taken for laboratory examination. The pus was not diagnostic on direct culture. Subcultures in broth revealed some short-chained streptococci and pneumococci. Because of the absence of organisms on direct culture, the possibility of anaerobic infection was considered. The sections of the mucous membrane showed it to be necrotic, polypoid and vascular with cells of chronic inflammation. There were some leucocytes, but more lymphocytes, and plasma cells. Some of the plasma cells had more than one nucleus. There were no epithelioid or giant cells. Smears made for tubercle bacilli were negative. The laboratory diagnosis of chronic sinus infection was made.

Postoperatively, 15 grains of sulfathiazole were continued every four hours for five days. The postoperative temperature ranged from 100° F. to 103° F. The lower lid became swollen and appeared for a while as though it might suppurate. X-ray examination of the orbital portion of the maxilla at this time revealed no bone pathology. Heat was applied externally. Limitation of motion of the eyeball disappeared in two days after the operation. The proptosis gradually was reduced. Pus in the nares disappeared on the first postoperative day and had not returned when the patient was examined four days ago. On the sixth postoperative day the temperature became normal, and recovery was uneventful. The boy was discharged from the hospital, recovered, on February 7, 1943, eighteen days after the operation.

CONCLUSIONS

I have reported a case of orbital cellulitis, secondary to a chronic maxillary sinus infection in a boy in whom the ethmoid was also involved. Pathogenic organisms probably entered the orbital cavity through an opening in the orbital surface of the right maxillary sinus, which apparently has closed prior to the operation, evidenced by the marked reaction about the orbital surface at operation. Conservative management had failed, but prompt radical surgical intervention, namely, a Caldwell-Luc operation and a transantral ethmoidectomy, together with sulfonamide therapy locally and systemically, accomplished the successful result.

185 North Wabash Avenue.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 7, 1942

THE PRESIDENT, DR. G. HENRY MUNDT, IN THE CHAIR

Anaerobes in Cranial Osteomyelitis

THOMAS C. GALLOWAY, M.D.

(Abstract)

The usual conception of cranial osteomyelitis on the basis of thrombophlebitis does not explain (1) why involvement of the maxilla and other compact bone may be so serious, or (2) why fulminating infections are more likely to follow minor interventions. The characteristics of such an invasion could be explained as the result of micro-aerophilic streptococci spreading along the periosteum, as Meleney demonstrated them burrowing along fascial planes; and minor procedures would make a covered focus in which these organisms would grow well. When these organisms are carefully looked for by proper technic in cultures from the depth or the advancing border of the disease, they have been found in nearly every instance of cranial osteomyelitis. Nine such cases are reported.

These organisms seem to be the primary invaders. That they are the important organisms is supported by the rather striking response of such infections carefully treated with activated zinc peroxide. Severe pain, tenderness, foul odor and induration seem characteristic. Anaerobes are probably also important in resistant infections of soft tissues of the head, such as Ludwig's angina.

DISCUSSION

DR. SHERMAN SHAPIRO: This is an important contribution because it shows that the anatomic explanation of Furstenburg and others does not give the entire answer. We have all seen infections of the frontal bone and the superior maxilla that do not always respond in the same way to treatment, regardless of the fact that they

look alike. It is very possible that Dr. Galloway has the solution. Recent military literature mentions that anaerobes have a predilection for bone. The spread along the periosteum may supply the reason why these cases of osteomyelitis behave so differently clinically.

My own experience with zinc peroxide is small. I tried it in one case which I reported last year. That patient got well with conservative surgery. Zinc peroxide did not achieve a cure, perhaps because of the inaccessibility of the infected bone. I used it in another case of stubborn antrum infection and was much impressed with the results. The discharge was noticeably less, the odor disappeared and a cure was obtained.

I think Mosher emphasized the importance of staphylococcus aureus in osteomyelitis. Is it Dr. Galloway's opinion that this organism lacks ability to produce damage or does he believe it can produce osteomyelitis by itself?

DR. GORDON H. SCOTT: Dr. Galloway suggests that infection in the cranial bones travels by way of the contiguous veins rather than through the diploic spaces. This does not explain the discrepancy in the speed at which infection travels in compact and diploic bones.

DR. DELBERT K. JUDD: I believe this subject will prove of great importance not only in the therapy of osteomyelitis but perhaps in other nose and throat infections. At least we cannot neglect to make a culture for anaerobes in spite of certain difficulties in technic. One wonders if persistent and frequent cultures might not eventually produce an anaerobe from a great many types of infection.

Dr. Galloway spoke of the cultures taken on the seven cases of osteomyelitis reported by Dr. Mosher and myself in 1933. It is true that the cultures were all positive for staphylococcus and in one case there was streptococcus as well, but now it would seem that these organisms may have been of secondary importance in the disease. Anaerobic streptococcus may have been present since anaerobic cultures were either not taken at all or not frequently enough to be of value. I do not remember that the presence or absence of anaerobes was considered. So far as the spreading of the disease through the bones of the calvarium is concerned it is definite that the process of retrograde thrombosis and thrombophlebitis plays an important role. In Dr. Mosher's seven cases, however, the inner table of the frontal bone or other involved bones of the calvarium were removed because of the frequency of extradural abscess with osteomyelitis. Furstenberg has shown that the infection spreads by way of the dura or endosteum. As the endosteum is stripped away from the inner table,

the bone is robbed of its chief source of blood supply, with the result that necrosis and sequestration take place.

DR. HOWARD C. BALLENGER: I wonder if Dr. Galloway has tried zinc peroxide in persistent maxillary sinusitis following extraction of an infected tooth which has eroded or ruptured into the sinus. Many of these bacteria are anaerobia.

DR. EDWIN J. BLONDER: Is this medication irritating to the eyes and, if so, is any protection used on the eyes when it is applied in the vicinity?

DR. GLENN J. GREENWOOD: I was interested in noting the relative times the cultures were taken in the course of the infection. Altemeier, reporting upon the bacteriology of war wounds, remarks that anaerobes present the first week have, by the third week, been replaced by secondary pyogenic invaders. He believes careful primary débridement to be one of the most effective prophylactic and therapeutic measures employed in successfully treating the anaerobic component of the infection.

DR. THOMAS C. GALLOWAY (closing): I do not think Dr. Shapiro's question concerning staphylococci can be answered with finality. In hematogenous osteomyelitis it seems able to do great damage by itself; yet in cranial involvement it seems more likely to be a secondary invader. Perhaps, however, it is responsible for the fulminating phase.

As to Dr. Scott's question regarding the method of spread of osteomyelitis, I believe Mosher holds the diploic venous spaces most important, and Furstenberg the dural veins. I should expect anaerobes to travel not in the vessels, but along avascular fascial planes, as for instance under the periosteum of flat bones. Perhaps again, when venous spaces are reached, the secondary staphylococci become important.

Zinc peroxide has not seemed irritating to the eyes when used near them. Closed lids kept it out rather well.

In just such a case of maxillary sinusitis as Dr. Ballenger mentioned, where anaerobic streptococci were cultured from an antrum secondary to dental infection, zinc peroxide was very effective.

Débridement, as mentioned by Dr. Greenwood, should be very effective both by removing necrotic infected tissue and by exposing any infection to aerobic conditions.

Posttraumatic Rhinoplasty: Secondary Correction of Deformities Involving the Septum and External Nose

SAMUEL SALINGER, M.D.

(Abstract)

A review of a large series of septal and nasal corrective procedures (more than 1000 cases) reveals that a large proportion of septal deviations are to a greater or less degree the result of trauma sustained some time previously. While it is generally believed that deviations of the septum are the result of errors in development, usually a disproportionate rate of growth between the septal structures and the bony cage surrounding the nose, careful analysis of many cases shows irregularities that could have been caused only by injury. Nearly all rhinologists have discovered that sooner or later the classic submucous operations of Killian and Freer are inadequate in coping with the situation; individual improvisations have crept into the technic, varying with the skill and the ingenuity of the operator.

In many cases correction of the septal deflection must be linked with correction of the external deformity. Unless a twisted nasal bridge is straightened, the submucous resection will often fail to give an adequate airway. Various procedures, such as the Metzenbaum technic, partial resection of the septum, hacking of the septal cartilage or removal of narrow strips to permit straightening of a curve, must be employed in order to preserve an intact septum and prevent collapse which frequently follows complete removal of the obstruction.

Various types of incision are shown to fit the individual case together with the procedure for straightening a deviation of the nasal bridge. As a rule the combined technic is preferred rather than separating the procedure into two steps. This makes for a better appraisal of the amount of septal tissue that must be sacrificed, and tends to conserve a greater amount than is usually the case if the septum is resected without regard for the external deformity.

DISCUSSION

DR. JOHN R. DELPH: I would like to ask whether it is advisable to attempt to correct the nose immediately after injury, or to wait until the initial edema has subsided, the soft tissue has been repaired, and the landmarks can again be seen.

DR. WALTER THEOBALD: I wish he would give his opinion as to the best age to operate on a patient with a deviated septum. He

showed a picture of a child on whom they worked for three or four years prior to operation. Opinions vary as to whether operation should be done at all before the age of puberty or before the age of 18. I have always discouraged operative procedures on the nose until the age of 19 or 20. However, some men operate in cases of severe deviation prior to that age to give an airway, and then complete the septal operation at a later age.

DR. GEORGE WOODRUFF: I wish Dr. Salinger would speak of his method of dealing with a traumatic saddle nose in cases he sees within a week or ten days following injury; that is, where a crushing injury to the nose would result in a traumatic saddle nose later on.

DR. SAMUEL SALINGER (closing): In reply to Dr. Delph, I would say if you wait 24 or 48 hours you are likely to have infection and that is the one thing that interferes with smooth healing. In the early correction of nasal injuries I am not worried about initial edema; it is not very bothersome. Most lacerations, if properly cleansed and sutured early and accurately, will heal with practically no scarring. Where suturing is inaccurate or the wrong suture materials are employed scarring will result, necessitating a secondary correction. Of course there are cases with massive injuries and shock in which it is inadvisable to do anything except control the hemorrhage and institute local antisepsis; yet if carefully sutured and replaced within 12 to 24 hours one may often duplicate the good results seen in cases that received early care. In one case, injured two months ago by flying glass, the entire nose and septum were completely cut across. I saw the patient within two hours of the time of the accident. No reaction had set in and I was able to sew up the septum, columella, nasal vestibule and external nose so that today one can barely see the site of the laceration.

In reply to Dr. Theobald, I would not do a submucous resection prior to the age of 16 or 17. However, this does not mean that one should not operate on the septum before this age. In the case of the child of 11 which was illustrated, I removed only the lower end of the septal cartilage; the remainder of the cartilage was infracted with the vomer and pushed back to the midline. There are cases of children whose lower end of the septum is buckled so as to block both nares. Such cases need relief, but not necessarily a submucous resection. One must contrive to so manipulate the cartilage and the supporting bone, if necessary, to provide breathing space without sacrificing the growing structure.

Concerning prevention of a saddle depression following trauma, naturally one makes every attempt to elevate and retain the depressed

cartilage and bone. Despite all precautions, however, a deformity will result from the absorption of small fragments and from scar contraction. The damage is not irremediable if one is prepared to insert a cartilage implant at a later date. I have done this on several occasions with excellent cosmetic results. One case was illustrated in which a compound fracture directly across the lower ends of the nasal bones resulted in depression which was subsequently filled out with a single cartilage isograft.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 4, 1943

THE PRESIDENT, DR. G. HENRY MUNDT, IN THE CHAIR

Primary Carcinoma of the Eustachian Tube: A Study of the Evidence of Its Occurrence

LAWRENCE J. LAWSON, M.D.

(This paper appears in full on page 377)

DISCUSSION

DR. JOHN F. DELPH: In studying my own cases and those in the clinic, I have been struck by the lack of early symptoms. Practically all cases were well advanced and presented multiple cervical metastases, hemorrhages, anemia, and in some cases neurologic signs of cranial invasion. From the case history one may often find mention of symptoms which might be construed as early signs of malignancy in the epipharynx, although not in themselves indicative of malignancy. Disturbances of the olfactory organ were noted in the complaint of fetor or complete anosmia. In practically all cases there were nasal or postnasal discharge and nasal blockage of some degree. Repeated small spontaneous nasal hemorrhages, recurring intermittently over a period of months, or one or two massive losses of blood requiring hospitalization were noted. Fullness of the ear and tinnitus, mentioned by Dr. Lawson, were practically absent in the series of cases studied. Disturbances along the distribution of the trigeminal nerve giving rise to mild pain were sometimes complained of. Martin describes this pain as a deep-seated, poorly localized distress which is usually persistent but occasionally paroxysmal. Other symptoms were a feeling of thickness in the soft palate, a nasal quality in the speech, disturbances of deglutition. In one case the first sign noted was a seropurulent discharge from the middle ear. Because of the tendency of these tumors to spread by contiguity and to form early metastases, the floor of the cranial cavity is frequently invaded, and the early signs then become neurologic in character. The oculomotor, the tro-

chlear and the abducens nerves are usually the first to be affected, causing diplopia; complete ptosis and ophthalmoplegia are later signs. Gradinego's syndrome was mentioned; this, when accompanied by a discharging ear, may cause confusion in diagnosis. Tinnitus and vertigo are often early indications of cranial invasion. Nausea and vomiting, with deglutition, speak for extension along the brain stem.

The above symptoms and signs have been gathered from a series of cases of proven lymphoepithelioma of the nasopharynx. They are not restricted to malignancies; a number of them are common to less serious lesions of the nose and throat. True, the prodromal symptoms are few and meager, but the occasional spitting of a small quantity of blood, nasal blockage or persistent nasal discharge calls for more thorough study of the nasopharynx than is generally given by the busy otolaryngologist. This can best be done by direct visualization by means of a palate retractor or the nasopharyngoscope.

I should like to report a case seen in consultation with the neurosurgical service, in which one of the early symptoms was referred to the ear:

R. C., a white man aged 39, was first seen because of the following symptoms: a diminution of hearing for five weeks, a sensation that the left ear was plugged shut for five weeks, right-sided periorbital pain for five weeks, a numbness of the skin over the right eye for two weeks, diplopia on looking to the right for three weeks, and difficulty in swallowing. A nasal discharge of mucopus had been present for five weeks. The neurologic examination showed a widened left palpebral fissure, a complete paralysis of the right rectus muscle, nystagmus on looking to the left, anesthesia of the right maxillary area, diminution of the right corneal reflex, and paralysis of the left side of the soft palate. There were deafness in the left ear and a nasal voice. There was no complaint of headache. Examination of the ears showed bilateral sclerotic membranes with an old perforation in the right. In the vault of the nasopharynx, obstructing the orifice of the left eustachian tube, was a mulberry-like mass of nodular tissue the size of a large olive, and extending from it across the roof was an apron of the same tissue. Large vessels were discernible. A biopsy report stated dense lymphoid infiltration with chronic inflammation. Because of the carcinoma-like appearance and the undoubted intracranial extension, the patient was started immediately on a course of radium treatment. A total of 300,000 mg. hours of radium was used in the form of external packs. Three and a half months after the beginning of treatment there was only a small nubbin of the tumor left, and most of the neurologic symptoms had disappeared. He is in

perfect health now after three years; there is no vestige of intracranial extension, and the mucosa in the epipharynx is smooth and clean.

This man undoubtedly had a visible nasopharyngeal tumor before he developed an intracranial extension, for the complaints were logical; that is, diminution of hearing and a feeling that the ear was blocked. The case illustrates the facts that the most prominent symptom, paralysis of the right lateral rectus muscle, may be contralateral to the primary tumor and also that the symptoms may be of short duration. Davis and Martin have previously reported this case history.

Dr. George Woodruff: I wish to mention two cases of carcinoma which originated about the eustachian tube orifice. The first was that of a boy aged 20. The tumor apparently originated in the fossa of Rosenmüller and was about the size of an olive pit. Symptoms were referred to the right ear and included blockage of the eustachian tube, tinnitus, loss of hearing and pain over the side of the face. There was no involvement of the cervical glands evident. The boy had had treatment elsewhere in an effort to relieve the obstruction. The tumor was discovered with the nasopharyngoscope. He was referred to Dr. Cutler about seven years ago and was treated with radium. He is alive and well today. There are no signs of recurrence; there is dryness of the nasopharynx.

The second case was more advanced. The eustachian tube orifice was covered by the tumor. Where it originated I could not say. There were demonstrable glands in the neck. As in the first case, the diagnosis was transitional cell carcinoma. This case also was treated by Dr. Cutler and is well today, about five years after he was first seen.

Dr. Arthur J. Coombs: While I have not seen many malignancies, I would like to mention two cases. The first, a man aged 46, had previously been operated on because of bleeding from the tonsil. When he came to me I discovered quite a large nasopharyngeal tumor coming from Rosenmüller's fossa. Biopsy showed this to be transitional cell carcinoma. There was adenopathy of the cervical glands on the same side, slight diminution of hearing, tinnitus, and later otitis media and pain. He was treated by the fractional method using x-ray, and apparently the tumor disappeared. About nine months later there was a recurrence. Further treatment was of no avail, although it was given over the glands of the body. He died of generalized metastases about two years after the tumor was first discovered.

The other case, a woman about 32 years of age, is now under our care at Illinois Research Hospital. When she was first seen last spring, there was a definite mass, about 4 x 8 or 10 mm. in size, protruding into the nasopharynx. Under x-ray therapy the mass disappeared rapidly. There was a recurrence this fall and she is now receiving further x-ray therapy. The radiologist advised against the use of radium. She developed convergent strabismus, involving that side, and an x-ray film shows the lesion invading the petrous portion of the temporal bone. In both these cases there was remarkable resolution of the process under x-ray therapy, but subsequently both patients developed a recurrence at the same site.

DR. GLENN GREENWOOD: I would like to ask how effectively the pain was controlled by radium. In the old days when there was pain from a nasopharyngeal new growth we injected the second or third divisions of the nerve with alcohol to control it. It was a most distressing symptom.

DR. LAWRENCE J. LAWSON (closing): The purpose of this paper is to emphasize two points: first, we as otolaryngologists should make careful and repeated examinations to find these inaccessible tumors early; second, these patients should be treated early and persistently by experienced radiologists working in close cooperation with the otolaryngologist. When this cooperation is lacking, therapeutic opportunities may be lost. Better types of radiation therapy are now available to produce better results.

The patient on whom I reported has not had the severe pain that is later produced in the invasive stage. We did not, therefore, have the problem of pain.

Otolaryngological Neoplasms—Clinical Report of Cases

L. B. BERNHEIMER, M.D.

(Abstract)

Several types of neoplasms including carcinoma of the lip, adamantinoma of the mandible, carcinoma of the floor of the mouth, carcinoma of the tongue, tobacco chewer's carcinoma of the buccal surface of the mouth, carcinoma of the tonsil, carcinoma of the larynx, metastatic melanoma of the larynx, chondroma of the larynx and carcinoma of the middle ear are reported.

Methods of treatment are discussed and clinical problems, such as prophylactic neck dissection for carcinoma of the lip, the choice between irradiation and surgery for noninfiltrating lesions of the larynx, and the end results of treatment of the types of tumors demonstrated are emphasized.

DISCUSSION

Dr. Samuel Salinger: Dr. Bernheimer has presented an interesting array of cases. In the main I can agree with everything he has said regarding therapy. Of course some of these cases are quite rare; adamantinoma, for example. I believe I have seen two cases, both in the superior maxilla. One is well after three years and the other was lost track of.

Melanoma is one of the most tragic conditions we have to deal with. I have never known of one being cured and the head of our tumor clinic, who has seen many of these cases, knows of only one that was cured. Since the tumor is radioresistant, the only chance for the patient is radical surgery. I am watching a young woman with a small lesion on the cheek which has been stationary for two years. When we first saw it, we advised a radical resection, removing part of the cheek and nose, but the patient objected. No one can predict the outcome, since it is well known that these lesions may remain quiescent for a long time and suddenly become active.

Dr. Bernheimer's experience with carcinoma of the tonsil is rather contrary to that of other observers. Most authorities who have access to large statistics feel that carcinoma of the tonsil is one of the most amenable of all malignancies about the head because of the lymphoid nature of the tumor and its bed. I will agree, however, that the presence of metastases diminishes the chances of cure. The few cases I have seen yielded promptly to irradiation and have remained free from recurrence for years.

Dr. Thomas C. Galloway: I think we all agree with the methods of treatment outlined by Dr. Bernheimer. Even ten years ago there was much disagreement as to treatment of any given case; now it seems rather well standardized. In the old days I coagulated many cancers of the tonsil with probably only one cure. Statistics show that about 24 to 28 per cent are curable by radiotherapy, which is definitely the method of choice.

Adamantinoma or ameloblastoma occurs more frequently in the mandible, but in the maxilla it is harder to control. It is a deceptive tumor; even though relatively benign it creeps along the periosteum

until it gets into the orbit or cranium and then the outlook is bad. I have one nine-year cure; three other cases were fatal. As to glands from tongue or cheek cancer, at a symposium of the American Radiological Association nearly everyone present agreed that block dissection was indicated early or when the first sign of malignancy develops. When glands are more than 1 cm. in diameter, the prognosis in most instances is grave.

The malignancy of melanoma is well known. Yet I saw a case 21 years ago which was treated elsewhere as a cavernous angioma, but was subsequently found to be a melanosarcoma. I saw this patient recently; he had multiple recurrences all over the forehead and in the nose, and yet he was carrying on his business.

Bronchogenic Carcinoma: Analysis of 125 Consecutive Cases

PAUL H. HOLINGER, M.D.

AND

H. JAMES HARA, M.D.

(Abstract)

Analysis of 125 consecutive cases of bronchogenic carcinoma examined at the Research and Educational Hospital and St. Luke's Hospital, Chicago, shows an increasing incidence with each succeeding year. The series consisted of 107 males and 18 females; 123 of the patients were white, one was a Negro, and one a Filipino. The oldest patient was 76 years of age, and the youngest 6 years of age; the greatest number of cases occurred in the age group of 55 to 59 years.

The significant symptoms noted were cough, hemoptysis, loss of weight and, in almost every case, chest pain or chest "discomfort." Pneumonia which became "unresolved" was quite common. Hoarseness was the first symptom noted in 7 cases; in 5 cases the left cord was paralyzed and in 2 the right cord.

In 98 cases, or 78.4 per cent, positive biopsy specimens were obtained bronchoscopically, and in 7 additional cases by exploratory thoracotomy, by chest fluid aspiration or from the sputum. In the majority of cases the bronchoscopy showed an intraluminal tumor, an irregular freely-bleeding bronchial wall, or a definite mucosal infiltration. However, in patients from whom it was impossible to

obtain tissue biopsy specimens, confirmatory information was obtained.

Inability to obtain tissue from a representative area is due to various factors. The tumor may be too far in the periphery or in an upper lobe. A tumor extending into the bronchus peripherally may compress the bronchus proximal to that invasion and prevent passage of the forceps to the intraluminal lesion. Not infrequently, too, a carcinoma so invades the mediastinum that satisfactory examination of the bronchi is impossible because of the "frozen" character of the tracheobronchial tree.

Squamous cell carcinomas were the tumors most often found, adenocarcinomas the least often, and undifferentiated carcinomas were between these extremes in frequency. The sites of the lesions were as follows:

Right main bronchus, 45; right upper lobe bronchus, 10; right lower lobe bronchus, 29; periphery (not visualized), 3; total on right side, 87.

Left main bronchus, 12; left upper lobe bronchus, 8; left lower lobe bronchus, 15; periphery (not visualized), 2; total on left side, 37.

Bilateral, 1.

Total, 125.

Examination of the American Board of Otolaryngology

The American Board of Otolaryngology will conduct its next examination at the Palmer House and the Illinois Research Hospital, Chicago, on October 6-7-8-9, 1943. All communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.